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THE ETIOLOGY OF CEREBRAL ABSCESS AS A COMPLICATION OF THORACIC DISEASE

J. LEIGH COLLIS, B.Sc., M.D., F.R.C.S.
BIRMINGHAM, ENGLAND

IN VIEW of the rather vague understanding generally prevalent toward many of the details of cerebral abscess it seemed necessary to examine it via an extensive survey of the literature and an adequate series of personal cases. For this purpose forty-six cases were collected from the Brompton Hospital, from the Birmingham United Hospital and from an E.M.S. Chest Center. The first forty-four of these were used for statistical purposes and details of these are shown in Table I.

The information from the various sources demonstrated certain points, the more important of which are now presented.

First, it was found that this is a condition preponderantly affecting the male sex, as shown in Table II.

Second, although no age group was exclusively involved, there was a tendency for people in late adult life to be most affected. This is tabulated in Table III.

Third, with regard to the frequency of occurrence of the condition, an idea is obtained from Nickerson's record of 10,502 unselected autopsies in which he found twelve instances of cerebral abscess associated with thoracic disease. With regard to the relation between this type of cerebral abscess and cerebral abscess from all other causes, the recorded figures agree fairly well to a proportion of about 14 per cent of all cerebral abscesses.

Concerning the frequency relative to the main primary conditions, Schorstein stated that 20 per cent of bronchiectatic patients eventually develop a cerebral complication. My figure of three cases in fifty-four

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TABLE I

CASE NO.	SEX	AGE	DEATH	PULMONARY CONDITION				BRAIN CONDITION			OTHER ABSCESSES
				NAME	TREATMENT	DURATION	BACTERIA	NUMBER PRESENT	SITUATION	DURATION OF SYMPTOMS FROM ONSET TO TERMINATION	
1	M	18	9/14/42	Bronchiectasis	Op.	13 yr.	Str.	1	Rt. temp.-occip.	12 days	Nil
2	M	29	10/12/41	Empyema	Op.	6 mo.	Str.	1	Lt. temp.	14 days	Nil
3	M	46	9/ 9/39	Empyema	Op.	12 mo.	Str.	14	Rt. frontal, rt. parietal, rt. occip. (2), rt. temp., lt. frontal, lt. parietal (2), lt. temp. (4), rt. cerebellum, lt. cerebellum	6 weeks	Perisphenic
4	M	13	2/23/39	Bronchiectasis	Nil	9 yr.		3	Both frontal, lt. occip.	19 days	Nil
5	M	31	4/28/39	Lt. lung abs.	Nil	5 mo.		3	Lt. occip., rt. occip., rt. temp.	4 days	Nil
6	M	39	5/ 9/39	Rt. lung abs.	Op.	19 mo.				3 days	Nil
7	M	41	1/ 8/39	Bronchiectasis	Op.	6 yr.	+ Str.	2	Rt. occip., lt. parietal	29 days	Rt. arm Rt. eye
8	M	44	11/ 7/38	Lt. empyema	Op.	15 mo.	+ Sterile	6	Rt. post horn. lat. vent., rt. below lower precentral, rt. below lower postcentral, lt. Parietal, lt. middle of precentral	5 days	Nil
9	M	26	9/29/38	Rt. lung abs.	Op.	18 mo.		Multiple	Both hemis., 1 cerebellum	7 days	Nil
10	M	47	9/12/38	Bil. lung abs.	Nil	6 wk.				4 days	Nil
11	F	41	9/27/37	Tb. empyema	Nil	6 yr.				7 days	Nil
12	M	53	8/11/37	Lt. empyema	Op.	3 mo.		1	Lt. temp.	3 days	Nil
13	M	19	5/ 8/37	Bronchiectasis	Op.	19 yr.		Multiple	Cerebral, cerebellum	2 days	Nil
14	M	17	Alive	Bronchiectasis	Op.	5 yr.					Nil
15	F	56	3/ 2/37	Rt. empyema	Op.	3½ mo.		4	Rt. front, parietal, occip., lt. temp.	5 days	Nil
16	M	32	11/12/36	Rt. empyema	Op.	9 mo.		Multiple	Cerebellum, cerebrum	6 days	Nil
17	M	36	11/25/36	Bronchiectasis	Op.	All life		1	Rt. frontal		

18	F	34	2/28/36	Rt. lung abs.	Op.	6 mo.	+ Sterile	2	Rt. frontal, lt. temp.	3 days	Nil
19	M	54	11/12/35	Lt. lung abs.	Op.	3 mo.		1	Rt. occip.	3 days	Nil
20	M	29	10/ 4/35	Bronchiectasis	Op.	29 yr.	Str.	Multiple	All over	3 days	Nil
21	M	56	10/ 3/35	Lung abs.	Nil	5 mo.				17 days	Nil
22	M	41	9/ 1/35	Lung abs.	Op.	10 yr.				6 days	Nil
23	M	54	3/12/25	Lt. lung abs.	Op.	8 mo.		2	Rt. occip., lt. occip.	16 days	Nil
24	M	39	10/10/34	Rt. lung abs.	Nil			4	Lt. cerebral h. (3), rt. cerebral h.	7 days	Nil
25	M	41	9/16/34	Lt. lung abs.	Op.	8 mo.		1	Lt. frontal	15 days	Nil
26	M	45	2/13/34	Lt. empyema	Op.	6 yr.				4 days	Nil
27	F	42	11/29/33	Lt. lung abs., empyema			Str.	Multiple	Both hemisphere, lt. cerebellum (1)	8 days	Nil
28	M	44	6/13/33	Rt. gang., empyema	Op.	4 mo.		2	Rt. parietal, lt. frontal	18 days	Nil
29	F	42	12/23/32	Rt. empyema	Op.	18 mo.				3 days	Nil
30	F	32	9/28/32	Bronchiectasis	Op.	14 yr.	Sterile	1	Rt. frontal	4 days	Nil
31	M	44	10/11/31	Rt. lung abs.	Nil	6 wk.		1	Rt. occip.	15 days	Nil
32	F	13	2/ 4/31	Rt. lung abs.	Op.	10 mo.		3	Lt. cerebral	5 days	Abdominal wall, buttock
33	M	50	11/27/30	Lt. lung abs.	Op.	18 mo.	Str. and B. (Gm. −)	1	Rt. frontal	3 days	Nil
34	M	31	6/ 2/29	Rt. lung abs.	Op.	11 mo.		1	Rt. frontal	10 days	Nil
35	M	22	1/ 2/29	Rt. empyema	Op.	18 mo.	Str.	1	Lt. occip.	--	Nil
36	M	42	1/15/28	Broncho pn., pul. tb.	Nil	5 yr.		1	Rt. parietal	26 days	Splenic abs.
37	M	55	7/10/27	Rt. lung abs.	Nil	8 mo.					
38	M	35	9/ 7/36	Gang., carc. lt. lung	Nil	3 mo.		1	Lt. cerebellum	15 days	Nil
39	M	19	1/ 5/26	Pul. tb.	Op.	3 yr.	Cocci (Gm. +)	1	Rt. frontal	8 days	Nil
40	M	41	2/ 4/23	Bronchiectasis, empyema	Op.	5 yr.		1	Lt. occip.	13 days	Spleen
41	M	44	10/10/21	Rt. empyema	Op.	8 mo.		1	Rt. occip.	8 days	Nil
42	M	41	1/18/20	Bronchiectasis	Nil	5 mo.		1	Rt. parieto-occip.	3 weeks	Nil
43	M	44	10/ 7/15	Bronchiectasis	Nil	44 yr.		1	Rt. occip.	--	Nil
44	M	40	4/15/12	Bronchiectasis	Nil	14 mo.		7	Rt. parietal, lt. parietal, lt. temp., lt. occip., lt. frontal, rt. cerebellum	25 days	Nil

TABLE II

	TOTAL	M	F
Charrier and Ferradou	51	43	8
Schorstein	19	16	3
Evans	17	13	4
Cohen	7	2	5
Nickerson	12	7	5
	106	81 (76%)	25 (24%)
Author's series	44	36 (82%)	8 (18%)

TABLE III

AGE INCIDENCE

YEARS	SCHORSTEIN	AUTHOR
2 to 10	10	0
10 to 20	7	6
20 to 30	12	4
30 to 40	15	8
40 upward	11	25

TABLE IV

FREQUENCY RELATIVE TO CEREBRAL ABSCESS OF OTHER ETIOLOGY

	PER CENT
Nickerson	20
Evans	11
Pitt & Moulonquet	16
Conchon	14
Eagleton	12
Meyer	13.4
Gull & Sutton	14
Bouchez	8

deaths from bronchiectasis at the Brompton Hospital between 1933 and 1938 only gives an incidence of 5.5 per cent. With empyema my figures show that the cerebral complication occurred in 1.9 per cent of cases and accounted for 16 per cent of the mortality of the condition. With lung abscess the corresponding figures were 4.5 and 20 per cent. It should be borne in mind that a large proportion of the empyemas in patients admitted to the Brompton Hospital were chronic; this bears out the point made by Buxton and White (1943) that brain abscesses only occur after chronic empyemas of more than three months' standing.

Fourth, all collections of cases show that, except for 4 per cent, brain abscesses in this group are secondary to bronchiectasis, lung abscess, or empyema. Pulmonary tuberculosis, carcinoma, pneumonia, and congenital heart disease are found in the remaining group.

The incidence of the three primary conditions is shown in Table VI.

Fifth, in considering the symptomatology, Evans and DeWitt have pointed out that the clinical picture of this type of cerebral abscess shows a marked difference in one respect from cerebral abscess of other

TABLE V

FREQUENCY RELATIVE TO PULMONARY INFECTIONS
(CASES AT BROMPTON HOSPITAL, 1933 TO 1938 INCLUSIVE)

	MALE	FEMALE	TOTAL	
Bronchiectasis:				
Inpatient	536	498	1034	
Died	35	29	54	
Cerebral abscess	3	0	3	(5.5%)
Lung abscess:				
Inpatient	184	59	243	
Died	46	8	54	(20%)
Cerebral abscess	9	2	11	(4.5%)
Empyema:				
Inpatient	235	81	316	
Died	28	10	38	(16%)
Cerebral abscess	4	2	6	(1.9%)

etiology. This difference is the rapidity with which the disease reaches a fatal termination.

This rapidity has a decided bearing on all aspects of the management of the case. It allows little time for diagnosis and makes any form of treatment unlikely to be successful. Schorstein found that death occurred within an average period of ten days, while my own series gave an average period of thirteen days from the first symptom to death. Except for this rapid progress of the disease, no other aspect of the clinical condition was found to be unexpected or to vary in any way from what is found in cerebral abscess from any other cause.

There were, however, three other clinical points which are considered worthy of separate mention. First, it is rare to find any other abscess present except the one in the brain. Evans states that in his seventeen cases there was only one instance of any other abscess being present and

TABLE VI

	TOTAL	BRONCHIECTASIS	EMPYEMA	LUNG ABSCESS
Evans	22	17	5	0
Schorstein	19	14	4	1
Charrier and Ferradou	51	19	14	12
DeWitt	1	1	0	0
Nickerson	12	0	1	11
Cohen	19	1	4	14
Flandin et al.	1	1	0	0
Wright	1	1	0	0
Roger and Alliez	2	2	0	0
Gordon	1	1	0	0
	129	57 (44.4%)	28 (22%)	38 (30%)

Author's cases:	
Lung abscess	16
Bronchiectasis	11
Empyema	11
Empyema and bronchiectasis	1
Empyema with lung abscess	2
Pulmonary tuberculosis	1
Carcinoma of lung	1
	43

in this one pus was found in both the spleen and right kidney. In my series five patients out of forty-three, where records were clear, had other abscesses present. In three there was a single abscess, while in the remaining two each had two abscesses. The sites of these additional abscesses are listed in Table VII, the main point of which is to demonstrate that no special area is selected.

TABLE VII

SECONDARY ABSCESS

SITE	NO.
Spleen or perisplenic	3
Abdominal wall	1
Buttock	1
Right arm	1
Right eye	1

The second point is the increased risk that surgery appears to bring. In the forty-four cases of my series, twenty-nine followed operative intervention. Further, in the eighteen-year period of 1912 to 1929, out of eleven cases, six followed surgery, while in the nine-year period, 1930 to 1938, there were thirty cases and in twenty-four of these surgical treatment had been instituted. During this latter period surgery had become more common and it will be seen that not only was the relative incidence of the surgical factor increased four times but also the total incidence was increased nearly six times.

TABLE VIII

SURGICAL RISK

CEREBRAL ABSCESS AT BROMPTON HOSPITAL	TOTAL	OPERATION	NONE
1912 to 1938	41	29	12
1930 to 1938 (9 yr.)	30	24	6
1912 to 1930 (18 yr.)	11	5	6

In conjunction with this it is to be noted that in Cases 2 and 26 of my series the condition followed immediately the dilating of a chronic sinus. The third clinical point concerned the type of primary disease present in the lung. This was marked by an absence of any acute conditions and further, the histories of the empyemas in my series showed an average length of ten months while the lung abscesses had an average duration of eight months. These figures do not include the cases of excessive duration, Cases 11, 22 and 26, which were of six, ten, and six years, respectively.

Sixth, from the pathologic aspect the condition is often spoken of as a spreading cellulitis in the brain but no evidence of this was found in my cases. The abscess cavities always had a clear-cut wall macroscopically, although microscopically there was little evidence of a fibrous reaction.

There is general agreement with Evans' statement that streptococci are frequently found in these abscesses but that a number are sterile. This naturally suggests that there is some specific affinity for brain tissue in the type of streptococcus found. Unfortunately there is a scarcity of complete bacteriologic investigation of these cases which is accounted for by the tendency for the abscesses to be opened for the first time at post-mortem.

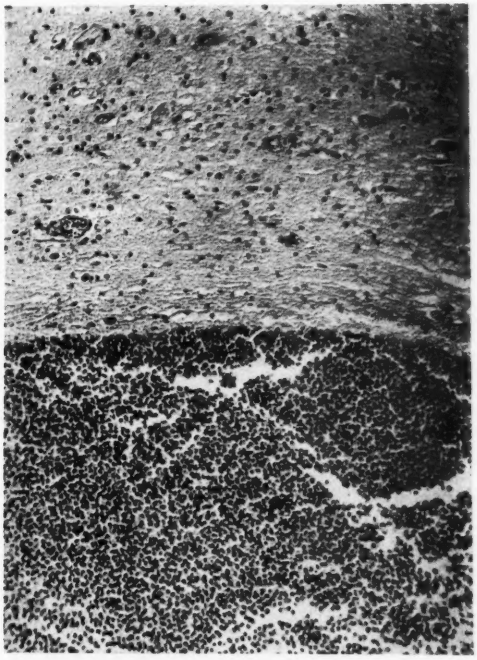

Fig. 1.—Low-power view of the wall of a brain abscess stained with hematoxylin and eosin. It shows the clear-cut edge as seen in many of these cerebral abscesses.

In this respect, however, the findings of McFarlan are instructive. He examined forty-eight patients who received operative treatment and of these, seven followed chest disease. In his investigation he laid special stress on the importance of anaerobic culture but was unable to find any association between the type of organism and the site of primary infection.

The occurrence of a proportion of sterile cases led McCordock to think that some organism difficult to find microscopically or on culture was responsible. He suggested that the spirochete might be the infecting organism and stated that he found it in two of the four patients he examined. This suggestion will be considered later but it may be

stated here that I was unable to find this organism in any of the five cases in which a search was made.

Falconer, McFarlan, and Russell have recently brought evidence to show that organisms readily die off in experimentally produced brain abscesses so that it would seem that no special significance should be attributed to this finding of a proportion of sterile cases.

Seventh, concerning the side and number of the abscesses there seems some evidence that the left side is affected a little more than the right, but the figures do not allow of any great significance being attached to this.

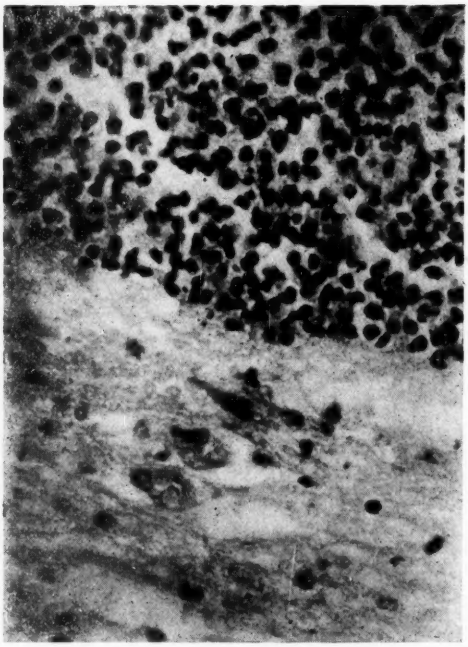

Fig. 2.—A high-power view of the same section which is shown in Fig. 1. The clear-cut edge is again demonstrated.

With regard to the number of abscesses present the figures in Table XI are produced.

It has been stated by Evans and more recently by Buxton and White that empyema is more liable to be followed by a single abscess than other conditions. This would suggest some fundamental difference separating the empyema group from those associated with lung abscess and bronchiectasis, but my figures show the same equal proportions following each of the primary conditions and further, the similarity shown by the same rapid clinical course does not allow of any such assumption being drawn.

TABLE IX

BACTERIOLOGY OF CEREBRAL ABSCESS

1. Author	15 cases	9 Streptococci 1 Streptococci + fusiformis 1 Streptococci + gram-negative bacilli 1 Gram-positive bacillus 3 Sterile (20%)

2. McFarlan — Organisms found in 48 brain abscesses (pus from one was sterile by films and culture)

	PUS CULTURE (33 CASES)	MIXED (14 CASES)	TOTAL (47 CASES)
Staph. Aureus	13	2	15
Str. pneumoniae	6	–	6
P. vulgaris	2	4	6
Str. pyogenes	4	0	4
Str. viridans	1	1	2
Nonhem. streptococci	2	–	2
Anaerobic streptococci	1	9	10
Unclassified streptococci	–	3*	3
Fusiformis	2	8†	10
M. tetragenus	1	0	1
Anaerobic diphth.	–	2	2
H. influenzae	1+	–	1

3. Seven chest cases from McFarlan's list

Empyema	Nonhem. Str.
Empyema	Unclassified Str. + fusiformis
Empyema	Unclassified Str. + fusiformis
Bronchiectasis	Fusiformis, Nonhem. Str.
Bronchiectasis + Empyema	H. influenzae

*Seen in films only
†Two strains of fusiformis in films only

TABLE X

SIDE OF ABSCESS

		TOTAL	LEFT	RIGHT	BILATERAL
Conchon		40	16	10	14
Schorstein		50	28	13	9
		90	44 (49%)	23 (25%)	23 (25%)
Author	Single	18	6	12	
	Multiple	10	18	18	

TABLE XI

NUMBER OF ABSCESSES PRESENT

	TOTAL	SINGLE	MULTIPLE
DeWitt	1	0	1
Schorstein	18	11	7
Parker	14	7	7
Cohen	2	2	0
Flandin et al.	1	1	0
Wright	1	1	0
Nickerson	12	8	4
Evans	17	11	6
Charrier and Ferradou	51	30	21
	117	71 (61%)	46 (39%)
Author's	35	18	17

Eighth, no part of the brain is immune from involvement. The cerebellum and the central part of the brain are rarely affected and most abscesses are found in the region of the junction between the white and the gray matter.

TABLE XII

SITE OF ABSCESS

SITE	SCHORSTEIN	CHARRIER AND FERRADOU	NICKERSON	TOTAL	AUTHOR'S
Frontal	9	5	1	15	15
Fronto-pariet.	4	7	0	11	
Parietal	9	4	4	17	10
Parieto-occip.	4	1	1	6	1
Occipital	4	3	1	8	15
Temporo-occip.	1	3	0	4	1
Cerebellar	2	1	2	5	4
Central	2	0	1	3	
Temporal					8

Ninth, with reference to the pathology of the primary condition in the chest, there would not appear to be any greater danger from disease on either the left or the right side. In seventeen instances of lung abscess of my series, eight were on each side one being bilateral, while with nine empyemas, five were on the left and four on the right side.

It was also evident that in the case of bronchiectasis and lung abscess in all my cases, the nature of the disease was severe. This has already been demonstrated by the long histories of the cases of lung abscess and it was also clear from the severity of symptoms and amount of sputum in the bronchiectatic patients. This would seem to be important as it has been suggested that in empyemas, the pleural changes are of no etiologic importance and that the cerebral complication would have occurred in any case as a result of the underlying lung condition. In nine cases of empyema where this lesion had been the main disabling factor from the start and not just a sequel to the lung abscess or bronchiectasis, it was found that there were only two patients with more than a trace of sputum and in one of these there was doubt as to whether it was not being caused by his coughing up of an empyema. In six cases in which post-mortems were performed, the lung tissue was found normal in three, purulent bronchitis was found in one, bronchiectasis in one, and in the last the lung tissue was normal except for an abscess the size of a pea. Having in mind the severe nature of the disease in the patients with lung abscess and bronchiectasis, it would not seem reasonable to give too great a significance to the relatively small amount of pulmonary disease in some patients with empyema. In fact it seems only logical to conclude that it is the empyema itself which is in some way causing the cerebral complication.

Tenth, the treatment of this condition has been most unsatisfactory. Only two recoveries have been recorded following surgical drainage. These patients of Wright and Barling were both of an unusually chronic

type. Very occasionally a patient will make a spontaneous recovery and one such case is included in the series recorded here.

CONCLUSION

In summarizing the information collected so far, it may be said to be divided into two parts:

First, there is a list of negative factors which by virtue of their equal distribution do not throw any light on the subject of the etiology.

1. Age
2. Side of brain abscess
3. Site of brain abscess
4. Infecting organism in brain
5. Number of abscesses present
6. Number of abscesses in brain following each of the primary conditions
7. The side of the primary condition in the chest

Second, there is a number of facts which seem to be most significant.

1. Involvement of male sex
2. The special clinical picture of this type of cerebral abscess
3. The essential similarity of all aspects of cerebral abscesses in this group whether they follow lung abscess, empyema, or bronchiectasis
4. The fact that the cerebral complication may follow disease in the lung or disease in the pleura
5. Only chronic disease or disease that has become chronic causes cerebral abscess
6. The increased danger following surgical intervention
7. The absence of abscesses in other parts of the body in association with the cerebral involvement

ETIOLOGY

In 1853, Virchow first drew attention to the close association between cerebral abscess and pulmonary septic conditions. He attributed the connection to the passage of a septic embolus from the lungs to the brain. This has remained the most popular theory ever since that time, although its inability to explain all the facts has led to many other views being put forward. The particular position of the lungs allows a septic embolus to reach the systemic circulation without having to pass a filter of the pulmonary bed. But this does not explain why such a large proportion of the emboli should arrive in the brain. Supporters of the theory have considered that the particular arrangement of the branches of the aorta might explain this, but if this were so the brain should be similarly selected in endocarditis of the left side of the heart. In seventy-six cases of this condition, Sperling found thirty-seven kidney, thirty-three splenic, and only seventeen brain abscesses. Further

experimental injection of infected material into the blood stream by Weed could only produce meningitis as a cerebral complication, while Kent and Blades have recently experienced a similar difficulty in producing a cerebral abscess. Another barrier to the acceptance of this theory was put forward by Watkins when he remarked that if this theory is true why do some cases not start with a syncope and signs of a cerebral embolism. This may be answered by saying that the emboli are too small, but there seems no reason why if emboli are formed in this way they should be confined to ones of particularly small size. It certainly would not conform to our knowledge of pulmonary emboli which are well known to vary widely in their size and the consequent severity of symptoms. Yet another stumbling block in the path of this theory is the place of empyema in the etiology. At best the explanation can only hold for the pulmonary circulation. This might be explained by saying the empyema is of really no importance and that it is the underlying pulmonary condition which is the real etiologic factor. Evidence has already been brought to show that this is not so and that the empyema itself must be accepted as being responsible.

In view of the unsatisfactory nature of this arterial embolic theory, Adam's paranasal theory will now be considered. In an effort to find a solution which would be more in accord with the facts it was suggested that these abscesses arose from infection of the paranasal sinuses in a similar way to cerebral abscesses following middle ear disease. This was supported in a paper by Watkins and they pointed to the frequent involvement of the frontal lobes and the common occurrence of upper respiratory infection in support of the theory. Watkins also pointed out that this would explain the absence of the apoplectic fit.

The evidence against the acceptance of this theory seems overwhelming. First this would bring the condition into line with cerebral abscess of other etiology and it has been shown that, if for no other reason, this cannot be accepted on account of the very much shorter clinical course of abscesses of this group. Second, though a considerable group of abscesses do involve the frontal lobes, there is a much larger group away from this area and for these the theory offers no explanation. Third, cerebral abscess from middle ear disease is always associated with suppuration in the ear of some severity. Conditions comparable to this were only encountered once in the thirty-six cases of my series in which autopsy was performed. It must be admitted, however, that the care with which this area was examined might not always have been great and a few cases might have been missed.

As this theory also breaks down on examination, Schorstein's views will be considered. He put forward the idea that the condition was due to the particular nature of the immunity reaction of the brain. He thought that the brain had a high natural immunity guarding it against acute infections although unable to build up a specific im-

munity following chronic septic states. It can hardly be believed that this is the whole explanation because if it be so, cerebral abscess should also be a complication of many other chronic septic states as ulcerative colitis and chronic osteomyelitis. Further, many of these patients, particularly those with empyemas, are almost cured at the time they develop the cerebral abscess and could not in the least be said to be in a chronic septic state.

As again no satisfactory explanation is to be found, attention will now turn to a review of McCordock's spirochetal theory.

Here an entirely new and most hopeful line was struck when it was suggested that these abscesses were produced by spirochetes. The fact that the original paper on this subject is only recorded in the *American Journal of Pathology* as having been read makes it necessary to rely on other authors quoting him for the statements he made. He drew attention to the common occurrence of spirochetes in septic pulmonary conditions and also for the preference shown by spirochetes for nervous tissue. In four patients he examined he found three were sterile and in the walls of two of them he was able to demonstrate with a Levaditi stain the presence of spirochetes.

This theory will certainly produce an explanation for many of the aspects of the case which have previously been so difficult, as for instance the special clinical course of this group of cerebral abscesses, but there are aspects of it which make it difficult to accept. First, it would seem that McCordock was placing too much importance on the finding of sterile contents in three cerebral abscesses. Figures concerning this point have already been produced to demonstrate that it is only a small proportion of abscesses that are sterile while the existence of some proportion of steriltiy is not surprising in view of the recent work of Falconer, McFarlan, and Russell, who have already been quoted as finding that organisms readily die off in experimentally produced brain abscesses. Second, although spirochetes do show some preference for nerve tissue, they do not give rise to the purulent reaction present in the abscesses of this condition.

In order to test the theory, Levaditi stain was used on the abscess wall in five cases as recommended by McCordock. It was not found possible to demonstrate spirochetes in any case. The microscopical appearances demonstrated by this method are shown in Figs. 3 and 4. It will be seen that the fragments of neuroglia take forms which are highly suggestive of spirochetes. It is not suggested for one moment that a man of the experience of McCordock fell into so simple a trap but it will be evident that, in the circumstances, examination of brain tissue by this staining method is difficult and productive of possible misinterpretation.

It will be seen that, up to this point, after putting aside pure arterial embolism, theories have been reviewed which lay special stress

on some local peculiarity of the brain. In considering this aspect, it is necessary to pay attention to the fact that streptococci are frequently present. It was felt that the findings would be extremely helpful if it could be ascertained that the streptococcus was of a type specific to brain tissue. There would seem to be sufficient evidence to negative this, but it does seem established that anaerobic or microaerobic streptococci together with other organisms of the same cultural characteristic find suitable surroundings in the brain. This group does not, however, appear selective enough to be the whole answer to the

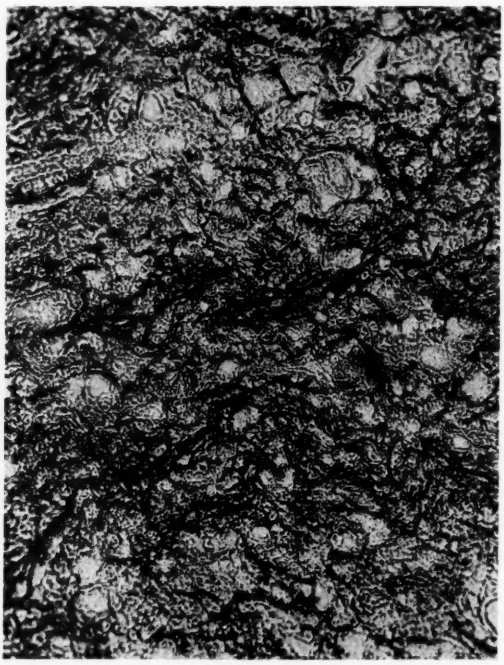

Fig. 3.—The brain tissue adjacent to a brain abscess stained with Levaditi stain. It is to be noted that McCordock stated that he found this appearance only in the brain tissue and not in the abscess pus. This alone is suggestive that it was brain tissue structure and not spirochetes.

problem and, further, it does not explain the increased risk after surgical intervention or the almost exclusive involvement of chronic or advanced cases. Against an all-important place being given to anaerobic organisms there are also reasons which are against any specific organism. This evidence is described later and is connected with the place of congenital heart disease and malignant disease of the lung in producing cerebral metastasis. Both of these strongly suggest a mechanical rather than a bacteriologic basis.

The possibility of some other organism not easily identified being responsible was also considered. The necessity of its being able to pro-

duce a pyogenic reaction makes the virus group unlikely candidates, but Dr. Marshall Findlay was kind enough to demonstrate to me that organisms of pleuropneumonia were capable of producing brain abscess by intracerebral injection. In view of this, specimens from two cases were examined by his methods, and in one case the actual tests were also carried out by him, but no positive findings were obtained.

In view of this it seemed that the only course left open was to review again the vascular connections between the chest and the brain.

Fig. 4.—A high-power view of another brain abscess stained by Levaditi stain. Again the appearance is suggestive of spirochetes.

In order to gain new light on this problem circumstances were looked for in which emboli could also reach the brain without passing the filter of the lungs. Congenital cardiac lesions seemed to present the necessary mechanical arrangement in view of the mixing of arterial and venous blood and the short-circuiting of the filter of the pulmonary bed. A paper on this subject by Abbott, Lewis, and Beattie (1923) recorded two cases of congenital heart disease complicated by cerebral abscess, and it was noted that both patients had an inflammatory focus present and that neither had any other evidence of metastatic spread except in the brain. Peacock was quoted in this paper as having said that cerebral complications were the commonest cause of death in congenital cyanosis. In a later paper by Baumgartner and Abbott, further comment was made on these cases and six more were discussed from the literature. They found the age range between 6 and 26 years and the sex distribution to be six males and two females. In 1940, Wechsler and Kaplan reported two additional cases and found twelve more from the literature. They confirmed the age to be usually under 20 years

and noted that the duration of the illness was unusually short, being on an average under ten days. This was a most interesting revelation as it has already been noted from the statistics of my cases as well as from the papers of Evans and DeWitt that this is a characteristic especially associated with cerebral abscess following thoracic disease. Both this fact and the sex distribution do suggest that there may be some close relation between these brain abscesses and the ones especially of interest in this paper.

Wechsler and Kaplan made another interesting observation in that cases of Fallot tetralogy were more likely to be followed by cerebral abscess than other forms of septal defect. This point was taken further by Hanna, in 1941, when reporting six cases of the same association of lesions with an additional seventeen collected from the literature. He found that of these twenty-three, a total of eleven followed the Fallot tetralogy with six complicating patent foramina ovale. The six other instances were associated with Eisenmenger's complex and other interventricular septal defects. The total result was, therefore, that seventeen cases were associated with ventricular septal defects against six complicating patent foramina ovale. This is an extraordinary finding in view of the more common occurrence of patent foramen ovale, and strongly suggests that there is some other factor at work apart from the simple mixing of the blood and short-circuiting of the pulmonary bed. A further interesting point from this paper was that the majority of the cases showed no septic focus from which emboli might have come.

The evidence seemed to suggest that the cerebral abscesses from bronchiectasis, empyema, lung abscess, and congenital heart disease were all in the same group but that the common connecting factor was not simple arterial embolism in the brain. There seemed to be a possible explanation of the real connection in the greater incidence found in lesions with a defective interventricular septum. The raising of the pulmonary blood pressure in these cases might be expected to cause an upset in the pulmonary vascular mechanics thus making the lung responsible for the secondary condition in the brain.

The conclusion that was drawn from this was that congenital heart disease does not give definite support to the arterial embolism theory but does suggest that some mechanical explanation is likely to be the true one.

This led to a more careful consideration of the view put forward by Eagleton, in 1922, that the condition was thrombotic in origin rather than embolic. This idea, taken in conjunction with Gardner's suggestion that the presence of a severe cough by raising venous pressure would reduce the vitality of the brain tissue, might appear a possible explanation. There are, however, some serious objections. First, some of the patients with empyema, as has already been stated, do not have a severe cough. Second, some conditions which give a very severe

cough as chronic bronchitis are never associated with cerebral abscess. Third, even if a thrombosis does occur this does not explain the presence of an abscess.

It seems necessary, therefore, not merely to find a mechanical explanation but to discover a mechanical explanation which includes a route for transference of infected material to the brain. It seemed also expedient to give more attention to the place of empyemas which appeared so frequently to be the stumbling block to the acceptance of any theory. With this in view other conditions were looked for, which might also spread to the brain. There is no other inflammatory condition, but carcinoma has associations in this direction which should not be overlooked. The frequent sequel of cerebral metastasis in carcinoma of the lung is well known although the exclusive selection of the brain does not occur as in the inflammatory conditions. Ochsner and DeBakey have recently published the figures given in Table XIII concerning the metastasis of 3,047 collected cases of pulmonary tumors.

TABLE XIII

	PER CENT
Regional nodes	72.2
Liver	33.3
Pleura	29.8
Lungs	23.3
Adrenals	20.3
Kidney	17.3
Brain	16.5
Heart and pericardium	12.7
Pancreas	7.3
Others	4.8

This list includes all cases of secondary involvement whether local, lymphatic, or blood stream spread, and it may well be that all these routes are not open in the same manner to the spread of an inflammatory process. However, if the secondaries in a particular organ as the brain are considered, the course by which they traveled is more likely to be the same. Figures were therefore obtained regarding the origin of cerebral metastasis (see Table XIV).

The significant point about these seemed not so much to be the leading position of bronchial carcinoma but the easy second place taken by carcinoma of the breast. Here is another chest wall structure to lend weight to the importance of empyema as an etiologic factor.

This made it appear all important to find some vessels which ran not only to the lung but also to the chest wall. The only ones fulfilling this requirement are the bronchial and intercostal veins which drain both the lung and chest wall into the azygos veins. Further veins from around the spinal cord also drain into the azygos system.

Harris (1941) has written a paper amplifying the very meager description of these vessels in modern anatomy books. They surround the spinal cord and communicate above with the venous sinuses of the

TABLE XIV

ORIGIN OF CEREBRAL MALIGNANT METASTASES

	NATIONAL HOSPITAL 1918 TO 1933	MEAGHER AND EISENHARDT	GRANT	ELKINGTON	HARE AND SCHWARZ	TOTAL
Total cases	72	40	43	17	100	
Bronchus	24	14	6	9	42	95(35%)
Breast	13	10	15	1	23	62(23%)
Gastrointestinal	7	2	2	1	8	20
Nasopharynx	6	2	4	1	0	13
Urogenital	14	2	4	-	5	25
Various and undetermined	8	10	12	5	20	55

skull and below with the intercostal and lumbar veins. They are valveless and as a result blood can readily flow in either direction, although gravity and differential pressure will usually direct this flow downward. Harris stresses the capacity of the system and cites as evidence a case reported by Trotter in which both internal jugular veins had been ligated without ill effect. Batson has also been interested in these veins. He was primarily concerned with the metastasis of carcinoma of the prostate to the brain. He made injections with radiopaque material into the dorsal vein of the penis of a cadaver and was surprised to find that a large part of the injection fluid instead of passing into the inferior vena cava traveled via the sacral region to the spinal veins. In the spinal veins he was able to trace the opaque substance right to the base of the skull. This led him to consider that these veins might play an important part in explaining paradoxical emboli. He also performed injections into veins from the breast and again noticed the great ease with which the medium passed toward the brain, and suggested that this venous system might also play an important part in the etiology of cerebral abscess from thoracic disease.

This hypothesis would seem to supply the very answer that the evidence so far is requiring and therefore in order to test further the possibilities of this venous system some injection experiments were undertaken on the cadaver.

In each case the body was lying flat on the back on the table, with a cannula inserted into an intercostal vein posteriorly so that no particular effect could be expected from gravity. Radiopaque fluid was then injected and its flow visualized both with a fluorescent screen and by radiographic plates. The injection was performed with a syringe and no accurate record of the injection pressure was made, although the delicate nature of these veins prevents any force being used. The radiopaque material was not the same in each of the five which were injected, but the most satisfactory results were obtained with 100 per cent silver nitrate to which was added one-tenth part of mucilage tragacanth. This was made with one part of tragacanth and two

parts of water. It would be anticipated that under these circumstances the flow would occur into the larger vessels and into the heart, and that the chances of its entering the vessels around the spine were small but reference to Figs. 5, 6, and 7 will demonstrate what actually occurs.

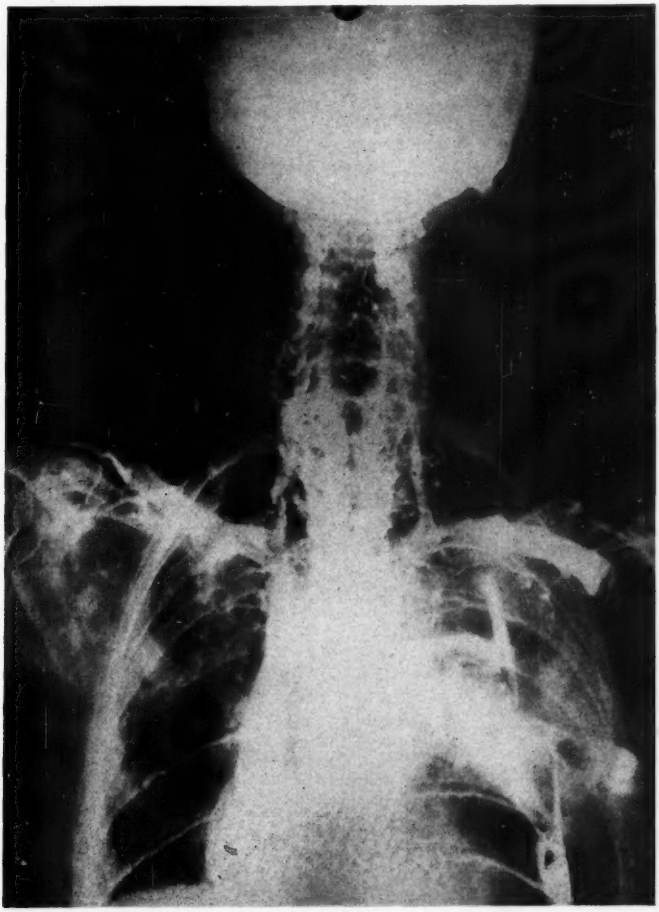

Fig. 5.—An intercostal vein on the left side has been injected and the cannula can be seen in position. The remarkable extent to which the fluid passes along the spinal veins to the skull is clearly shown.

In order to test the possibilities of this route further, survival animal experiments were undertaken. The cat was chosen as an animal which withstands surgical intervention although it is not naturally susceptible to cerebral abscess. The experiments were carried out in ten cats by performing a right thoracotomy and injecting into the azygos vein. A mixture of equal numbers of streptococci and staphy-

lococci from empyemas and lung abscesses was used to which was added an equal number of *Bacillus coli* because of the cat's susceptibility to this organism. To this was added India ink in an effort to produce particulate matter of a similar nature to small emboli. In

Fig. 6.—Again an intercostal injection has been performed but this time on the right side. A similar filling of the spinal veins is seen and again it is remarkable how relatively little of the fluid can enter the larger vessels. This is, of course, due to the action of the valves.

each case the arch of the azygos was clamped during the injection and for two minutes after it. This was done in order to reproduce the conditions of raised venous pressure during coughing or straining. Except for the first animal the cats stood the operation well and the only local complication was an empyema in one case.

Unfortunately, I have to report that I was unable to produce a cerebral complication in any case. One cat developed a staphylococcal mastoiditis while three cats, after making complete recoveries, died between seven and fourteen days later for no reason that could be found at autopsy. It is possible that the cerebral lesion was too small to be found but no evidence for such an assumption could be obtained.

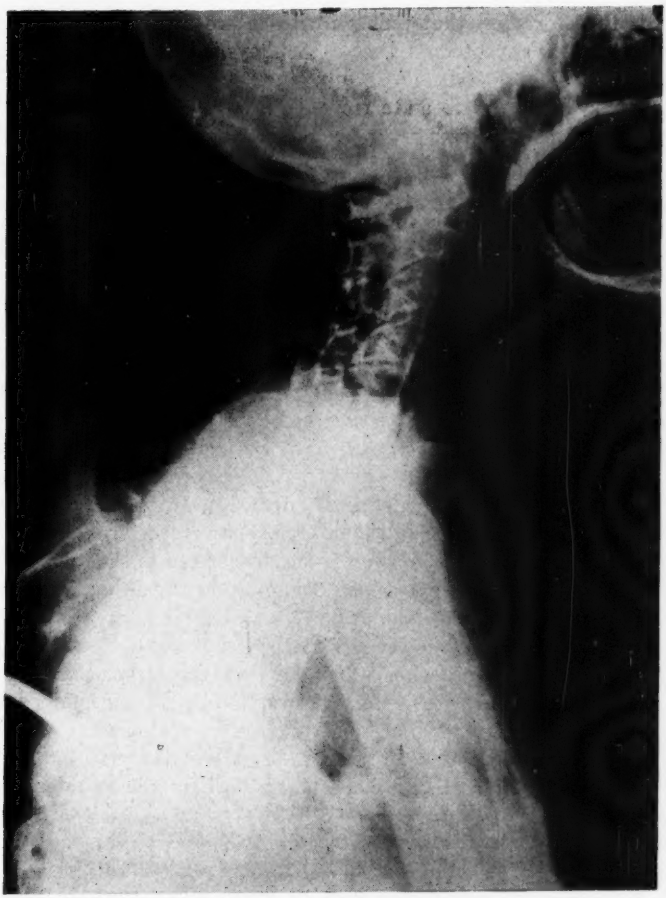

Fig. 7.—A lateral view of the same as that shown in Fig. 6, with the radiopaque fluid passing up behind the bodies of the vertebrae and around the spinous processes to the skull. In each case 40 c.c. of opaque solution were used.

There are obvious disadvantages for animal experiment in this condition. Organisms virulent to man may be easily killed by the cat while the circumstances of free organisms in the blood stream are probably not similar to the state in man. Most important of all is the fact that the cat walks on all four limbs and never lies down on

his back. As a result of this, gravity will never have a chance of encouraging a reverse flow in the spinal veins. Lastly, it was noted in the experiments that the anastomosis is so good between the intercostal and the superior vena cava that even with the azygos clamped close to this vessel, the azygos vein did not become distended, while the India ink injected appeared immediately in the lung tissue. This was timed and found to be less than one second.

It is therefore felt that unless facilities are available for these experiments to be repeated on apes or monkeys it is unlikely that helpful results will be obtained.

It is now proposed to state more definitely the way in which I believe that these abscesses are produced and especially how the spinal veins fit into the picture.

Thrombosis starts close to the inflammatory process in the lung or pleural cavity and gradually extends from the capillaries to larger venules. In order for it to spread to the vessels of the chest wall it is necessary for the adhesions between the lung and the chest wall to have become vascularized. Later the thrombus involves the walls of larger veins without completely occluding them and later still the thrombus begins to break up and form emboli in the blood stream. Most commonly these will affect the intercostal veins because the distribution of the bronchial veins is relatively limited. The thrombi will then be carried to the azygos vessels, from where in most cases they will be carried to the superior vena cava and then will be caught up in the pulmonary bed without ill effect. If, however, the thrombus becomes detached when conditions favor a reversed flow in the spinal veins, then it may enter this system and be carried to the brain. These conditions will be present while coughing or straining at stool, or when the patient is lying flat on his back. Having reached the brain the thrombus will become lodged at the venous end of the capillary bed and will in consequence affect a relatively larger area than an arterial embolus of the same size and will also be a certain amount nearer the surface than the similar arterial embolism. When the embolus has reached this site it will be more likely to be followed by abscess formation if it is infected with anaerobic or microaerobic organisms.

Evidence has already been given of the potentialities of the spinal vessels and reference to Figs. 8, 9, 10, 11, and 12 will demonstrate the process of thrombus formation and its spread through the chest wall to the intercostal veins.

In conclusion, an effort will be made to show how this theory explains the facts of the case. First, the exclusive involvement of the brain is easily understood. Second, the increased danger of the condition as a result of operative interference is due to the presence of an infected sinus in close proximity to an intercostal vein and its tributaries. If the drainage involves more than one rib as is often the

case in lung abscesses, the danger of cerebral complication might be expected to be increased.

Third, the point that acute pulmonary conditions do not give cerebral complications is explained from the fact that these will have little chance of access to either the intercostal or bronchial veins, since the bronchial veins only drain the main bronchi while the chest wall will not be affected to any severe extent.

Fig. 8.—A section taken from the wall of a lung abscess adjacent to the chest wall. A number of capillaries are shown. One of these particularly, at 8 o'clock below the center is thrombosed.

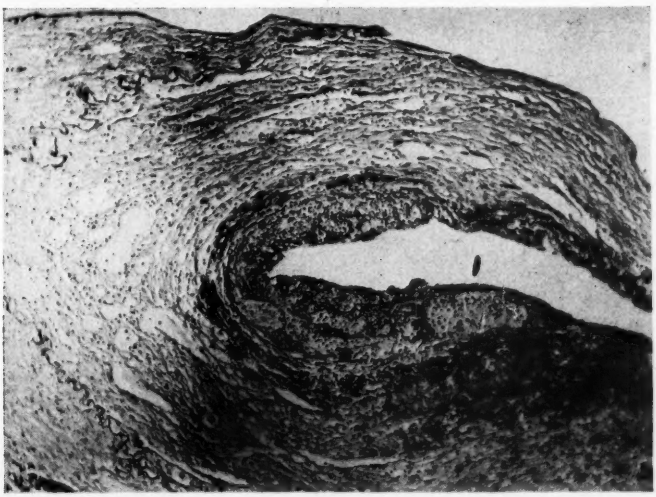

Fig. 9.—A vessel further away from the abscess but still not in the chest wall, which shows the formation of thrombus in its wall.

Fourth, the special importance of chronic conditions is due to the fact that it takes considerable time for the pleural adhesions to become vascularized so that a thrombotic process can spread across them to the intercostal veins.

Fig. 10.—A vessel similar to that shown in Fig. 9, but here the thrombus has broken up and is lying free in the blood stream.

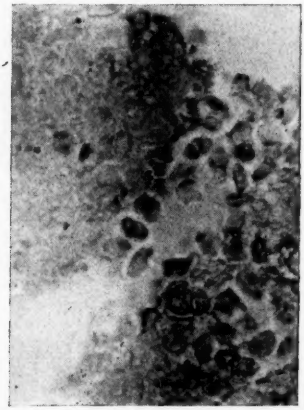

Fig. 11.—A high-power magnification of a portion of the free thrombus in the vessel shown in Fig. 10. In the center will be seen a pair of gram-positive organisms.

Fifth, the fact that empyema only causes this condition when it is of considerable duration from surgical drainage is explained partly because time will be necessary for the new area of the chest wall to become affected and partly because when the patient is recovering and

is up and about the increased respiratory movements will aid the dislodging of thrombi.

Sixth, lung abscess and bronchiectasis are only able to produce the condition when they are of severe degree or have been treated surgically because only under these circumstances can the intercostal or bronchial veins be affected.

Seventh, the different clinical history of cerebral abscess from chest disease and cerebral abscess from other causes is easily understood in cases in which the latter is due to direct extension, while in others the slightly different anatomic position of an abscess situated at the venous rather than the arterial end of the capillary bed may be significant.

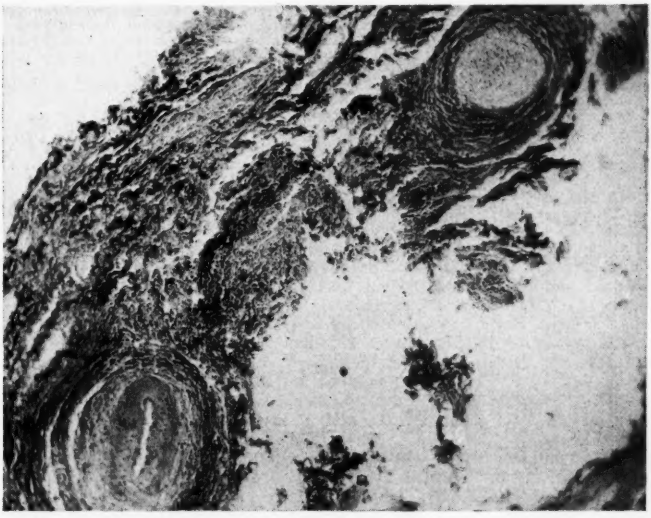

Fig. 12.—Showing two vessels in the chest wall and some intercostal muscle between them. The small artery is still patent, but the small vein is thrombosed. These are not the main intercostal vessels which were much bigger and in this patient were not thrombosed.

Eighth, the etiologic importance of both pulmonary, pleural, and chest wall structures can easily be understood, in view of the common reservoir that both intercostal and bronchial veins find in the azygos vein.

Ninth, the occurrence of a similar spread to the brain in carcinoma of the lung and breast can be explained.

With regard to the clinical importance of this theory it is suggested that, when a rib resection is performed for surgical drainage, the intercostal veins should be occluded so that there will be no chance of a process of thrombosis spreading to them.

Another point of prophylactic importance which has been demonstrated is that in empyema and lung abscess the condition only follows

chronic cases. This allows good reason for hope that with empyema at least modern treatment of the primary disease will materially reduce the incidence of the cerebral complication.

SUMMARY

A careful review has been made of all previous proposals concerning the connection between thoracic disease and cerebral abscess. Considerable evidence has been given in favor of the view that infected material from the chest is carried to the brain via the spinal veins and that, if this material is infected with anaerobic or microaerobic organisms, it is then more likely to result in abscess formation.

I wish to thank the members of the staff of the Brompton Hospital, London, and the Birmingham United Hospital for allowing me to use data on their cases. I also am most grateful to Mr. W. J. Pardoe of the Anatomy Department of the Birmingham University for the photographic reproductions.

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HYDATID CYSTS OF THE LUNG

LOUIS R. DAVIDSON, M.D.
NEW YORK, N. Y.

THE discussion of this subject, hydatid cysts of the lung, is based upon a personal operative experience of four cases. The presentation is prompted by the fact that in the United States this disease which is now uncommonly met with, unfortunately will be found to be much more frequent in the returned soldier. In addition, these four cases revealed sundry symptoms and signs and the operative procedures of necessity varied. Obviously, this cannot be a general or a complete study of hydatid disease of the lung. I was assisted in my understanding of this malady by referring to various publications and papers.

The lung, after the liver, is the organ of the body most frequently infected. Hydatid cysts of the lung were clinically known to the ancients. In modern times considerable impetus to the study of this tapeworm disease of man has been afforded by the work of Dévé, the eminent French authority, by the studies of Harold Dew of Australia, and by the writings of José Arce, the Argentinean surgeon. These include, among other matters, the different manifestations of hydatid cyst, such as metastatic and secondary hydatid disease. The geographical distribution of this condition "is dependent on its incidence and distribution in reservoir hosts, particularly sheep, cattle and pigs."[490]

Native cases are uncommon in this country but they are far from infrequent in sheep-raising countries, such as Argentina, Uruguay, New Zealand, Australia, Italy, Syria, Greece, and Algiers.

The development (Fig. 1) of the cyst in man is readily understood if a few brief moments are given to the study of the evolution of the taenia and the larva. Of course, where there are sheep, there are dogs. The infected dog (also the wolf and jackal) is the definitive host; in its small intestine worms or taeniae (echinococcus granulosus) are found. The intermediate or reservoir hosts are many, but most commonly infested are the sheep, pigs, and man. An essayist might say that it is no news when a dog bites a man; he may also say that in modern times a dog does not eat a man. Because of this fact, one may relate that man is an "accidental host as far as the further biological development of the parasite is concerned; human infestations represent a dead end" (Dew). Man then does not take part in the continuation of the cycle. The untainted dog is infested by ingesting the organs of animals, usually the sheep, which have been afflicted with fertile hydatid cysts. We note that the hydatid cyst in man is the result of the development of an ovum, or ova, in an organ, or organs, of man. The ovum has its origin in the

Read at the Twenty-fifth Annual Meeting of the American Association for Thoracic Surgery, Chicago, Ill., May 5 and 6, 1944.

Taenia echinococcus which resides as a parasite in the dogs' intestine. This worm is very small (Fig. 2). Its length is about 0.5 cm. A chain of segments forms the entire tapeworm. In is composed of a head, neck, and two segments or proglottids. The terminal compartment is gravid

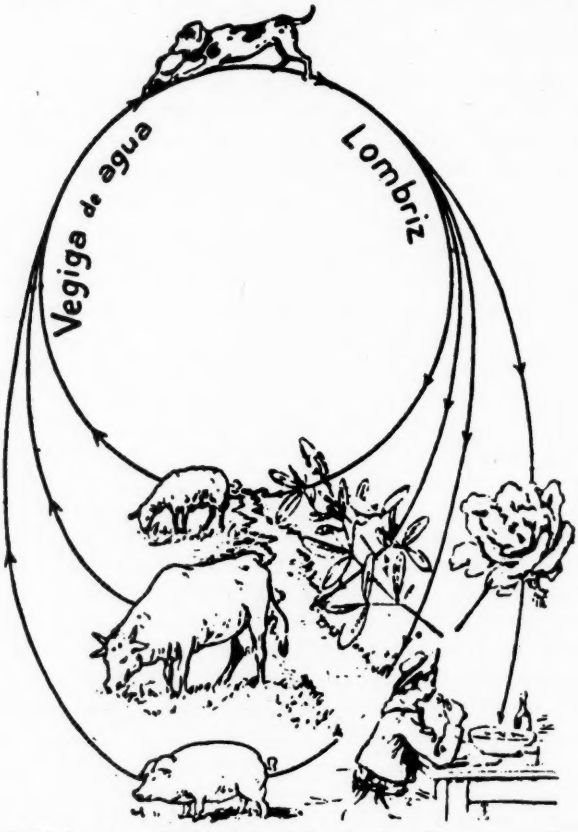

Fig. 1.—Depicting the natural history of the *Taenia echinococcus*. (From Vegas, M. H.: Hydatid Cysts of the Lung in Children, Buenos Aires, 1928, Imprenta Lamb y Cia. Ltd.)

Fig. 2.—*Taenia echinococcus*, showing all segments.

and it is from this, the broadest segment, that eggs are discharged. The head is provided with four suckers and is armed with many hooklets by which the taenia fasten themselves to the intestinal mucosa of the dog. An infested animal contains many hundreds or thousands of worms;

in turn these taenia discharge into the feces innumerable ova which gain entrance into man (the intermediate host) through contaminated water or vegetables.

When the egg (Fig. 3) lodges in man's stomach the gastric juice digests the external coat thus freeing the hexacanth (six-thorn) embryo. Subsequently, it migrates through the gastric wall or the duodenum entering the venules and then lodging in the first barrier, the liver. If this shelter is hurdled the embryo passes through the inferior vena cava into the right side of the heart and then is filtered out of the blood stream by the lung.

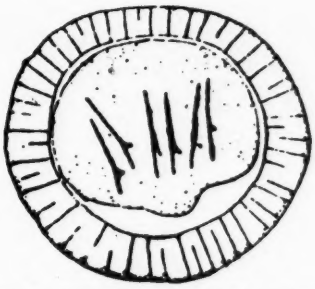

Fig. 3.—Hexacanth (six-thorn) embryo.

Let us consider that it remains and develops in this organ (Fig. 4) and there assumes what is called the larvated stage. The embryo in growing empties its center. The vacuolation continues so that at a certain point one finds a small vesicle which contains fluid. The expanding embryonal wall is composed of two zones; the inner nucleated wall (germinal layer) and the outer nonnucleated wall (laminated layer). From the former wall masses of cells which become vacuolated protrude into the vesicle's cavity. These capsules form the proliferative vesicles or brood capsules which in turn give rise to the scolices. These are the heads or the source of new worms. Most of these capsules and scolices are pinched off the germinal layer and sink in the hydatic fluid *(aqua de roca,* crystalline fluid), becoming what has been called "hydatid sand." The fluid-containing bag, composed of two layers and hydatic sand, is the hydatid vesicle. As the vesicle (Fig. 6), which is the parasite, grows, the organ in which it resides reacts to its presence forming a perivesicular layer or adventitia. Obviously, this is formed by the host. The connective tissue layer which surrounds the hydatid vesicle forms, with the latter, the hydatid cyst. The adventitia embraces the hydatic vesicle. However, it is not united to it. This holds true in the simple cyst. The adventitia bears the same relationship to the vesicle that the banana skin has to the fruit. Let us say this cyst developed in a sheep instead of man; further imagine that the infested organ of this slaughtered animal is eaten by a dog. The scolices are ingested and enter the small intestine where they become attached to the mucosa developing

into *Taenia echinococcus*. Obviously, the entire cycle is repeated. Nothing will be mentioned about daughter cysts which occasionally develop except to indicate that they are usually the result of a mechanical, infectious, or toxic influence and usually they are biologically sterile, that is to say, they are without scolices or proliferative vesicles. On the other hand, a fertile cyst, if ruptured or aspirated, may discharge scolices into the surrounding tissue, thus causing the origin of new cysts which are known as secondary cysts (secondary hydatidosis). This leads to the obvious truism that when operating one must not

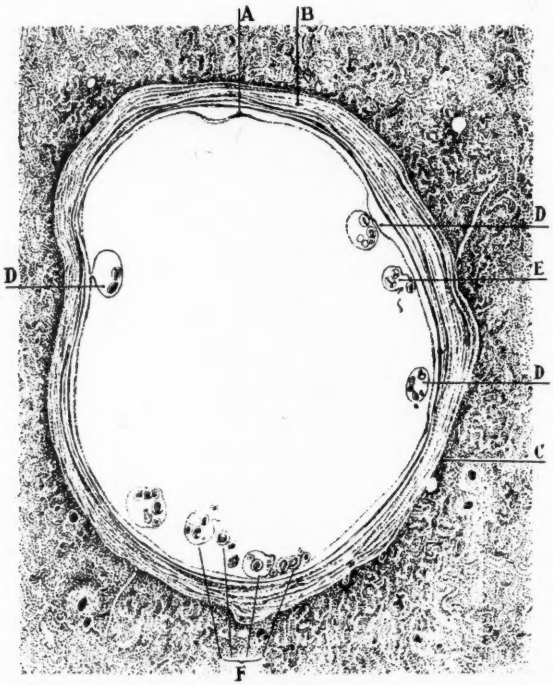

Fig. 4.—*A*, Germinal Layer, *B*, Laminated Layer, *C*, Adventitia, *D*, Proliferative vesicle, *E*, Scolices, and *F*, Hydatid sand. (From José Arce: Arch. Surg. 42: 3, 1941.)

permit any of the hydatid fluid or hydatid sand to contaminate the incisional or operative wound. In addition, for this reason, among others, one should never aspirate a hydatid cyst except at the time of operation. Again, if the primary cyst erodes into the pulmonary vein, metastatic hydatidosis results.

The fluid and the membranes in the simple cyst are bacteriologically sterile. Of course, cysts without scolices and brood capsules are biologically sterile and are called acephalocysts. Simple hydatid cysts may assume a new phase and become complicated. When this occurs,

the adventitia becomes infected thereby altering the integrity of the germinal and laminated layers along with the contents, with resulting death of the vesicle. In consequence of this, the cyst is septic and the hydatid vesicle virtually falls apart. The contents are necrosed and fragmented (see Case 3).

In the lung the parasite develops not only into a hydatid vesicle with its surrounding adventitia, but also the latter is covered by a thin concentric layer of atelectasis. The enlarging cyst (Fig. 5, *A*) impinges upon a bronchiole whose wall may contact, through an adventitial defect, the outer surface of the laminated layer. As the cyst increases in size the bronchiole is eroded, thus permitting the perivesicle space to communicate with the bronchiole. Now through various acts such as coughing and straining, air insinuates itself between the "banana skin and

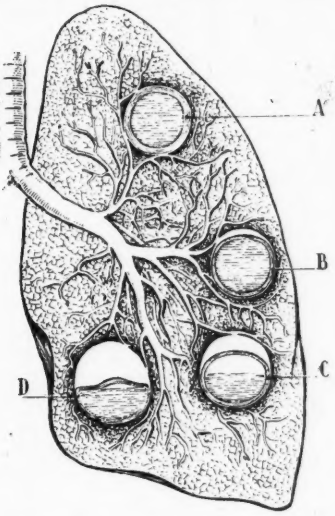

Fig. 5.—*A*, Hydatid cyst of lung, *B*, Perivesicular pneuma, *C*, Double-dome Arch (Cumbo's sign), and *D*, Camalote formation. (From José Arce: Arch. Surg. 42: 11, 1941.)

fruit." If the membranes tear, fluid enters the bronchiole and the act of coughing is stimulated. Then more air surrounds the vesicle causing the perivesiculum pneuma (*B*). In most instances further changes are noted. If a larger opening occurs in the membranes, air enters the vesicle and a double-dome arch (*C*) Cumbo's sign is produced. Finally, when the echinococcus membranes sink into the cavity and float upon the remaining fluid, the camalote (*D*) formation is noted. This was first described in 1924.* Further interesting, though infrequent, changes occurring in the complicated cysts may be reviewed in many of the articles mentioned in the bibliography.

*See Annals 20: 276, 1943

It is necessary, for the understanding of the symptoms and signs, to recognize the difference between simple and complicated cysts which may be either peripheral or central. The simple or ordinary cyst generally is silent. Cough is usually the first symptom and, of course, is nonproductive. Occasionally, there is companion chest pain. Later the cough may be productive and accompanied by hemoptysis. Later, when the cyst enlarges, dyspnea may manifest itself. This symptoma-

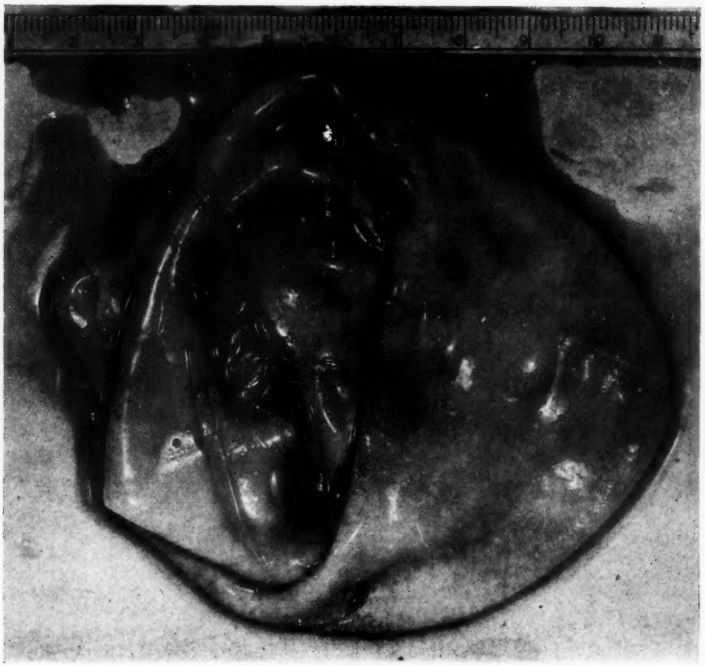

Fig. 6.—Membranes extracted from patient in Case 1.

tology does not reveal the possibilities of an exact diagnosis. The physical examination often suggests the diagnosis, but of course it is not unmistakable. "In countries with echinococcus-infested zones, the place where the patient lives is of the utmost importance, in cases of doubt.[491]

Diagnosis is difficult at the best. Laboratory methods are quite valuable. Yet these procedures are not unequivocal. Eosinophilia is not present in dead or suppurating cysts. Even in intact cysts the positive blood picture is not always present. Writers differ, yet one may state that eosinophilia is present when the percentage of eosinophiles is over 4 per cent. Of course, other conditions produce this picture. If a high eosinophilia count is obtained one or the other of the biologic reactions, which are about to be described, should be present before a diagnosis is made. The complement fixation test (Weinberg) is not invariable. Of

all tests the intradermal one of Casoni is most reliable although it, too, is not constant. Certain diagnosis is made only when hooklets (Figs. 7, 8, and 9), scolises, or laminated membranes is found in the sputum. Yet, in complicated cysts one can make a positive diagnosis when a characteristic x-ray picture is found noting the perivesiculum pneuma, Cumbo's sign, or camalote formation. In Case 3, a preoperative diagnosis was made simply upon the basis of such an x-ray.

Medical treatment is unavailing. Puncture of the cyst, whether for the purposes of diagnosis or treatment, is mentioned merely to be condemned. It follows then that surgery is the only treatment. What cysts should receive intervention and when ought they be operated upon? There is some difference of opinion. However, one must ask in what group is the cyst? Is it simple or complicated? Central or peripheral? Simple cysts offer no symptoms; occasionally there is a slight unproductive cough with some blood spitting. If the parietal pleura is contacted by the cyst there may be some pain. Complicated cysts frequently reveal

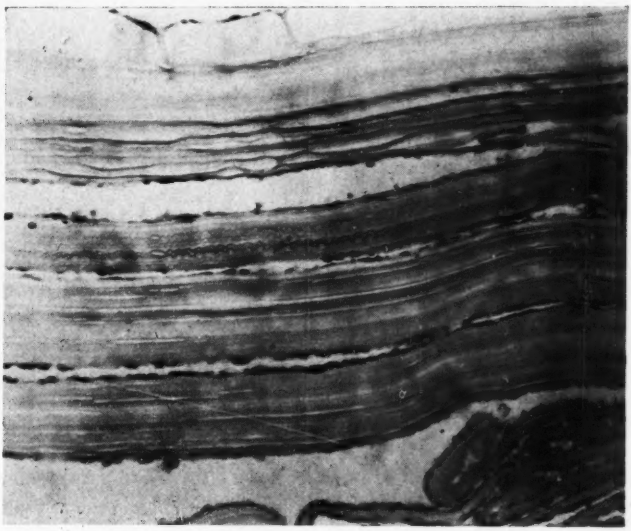

Fig. 7.—Germinal layer and laminated layer (Case 1).

pathognomonic x-ray signs. As in a putrid lung abscess, the presence or absence of pleural adhesions determines the type of surgical therapy. Regardless of the presence or absence of pleural synechia, the essence of treatment is identical; the chest wall is opened, the cyst wall is incised, and the membranes are removed. A one-stage operation in the absence of adhesions is neither simple nor without a definite risk, particularly that of pneumothorax. Central cysts should not be attacked unless they are draining poorly and are accompanied by fever. Even then caution is suggested.

The chest wall opening is made over the site of the peripheral cyst which previously has been revealed by x-ray. Some authors advocate a preoperative pneumothorax to determine the presence or absence of adhesions. With the presence of adhesions between the visceral and parietal pleura definitely established, and after the parietal pleura has been exposed, the cyst is entered and the membranes and contents are removed. If when the parietal pleura is exposed, the lung is found unadherent, the surgeon may produce adhesions by packing the wound with gauze (see Case 1). About two weeks later the packing is removed and adhesions will be noted between the visceral and parietal pleura.

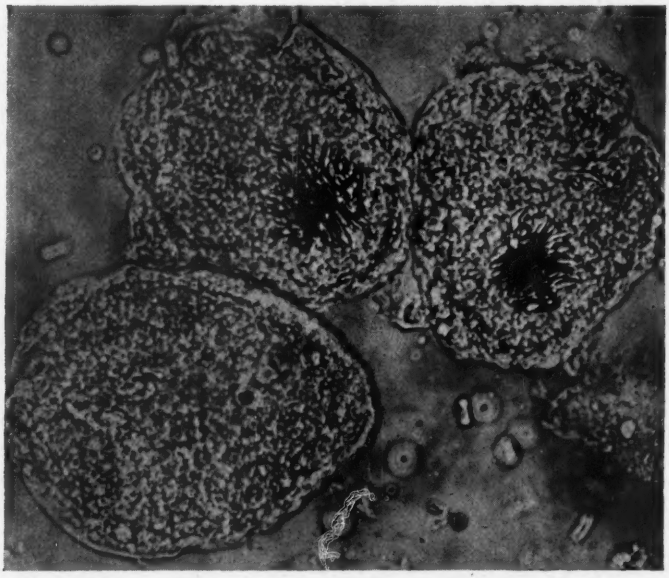

Fig. 8.—Scolices with cupping (Case 2).

Then the cyst is opened and emptied. On the other hand, a one-stage operation is conducted by projecting the lung through the wound in the parietal pleura. This is accomplished through the use of a tight-fitting gas mask, at the appropriate moment the pressure in the bag being increased to about 10 cm. of water, positive pressure. Another type of one-stage operation for the unadherent lung is performed by transfixing the lung, pleurae, and part of the chest wall with sutures outside of the cyst area. Thus, the lung is held to the chest wall, preventing a large pneumothorax.

The cyst itself is treated in different ways. Of course, the wound should always be meticulously draped before the cyst wall is opened. After an exploratory needle has been thrust into the cyst, fluid is withdrawn. Some complicated cysts have no fluid but this is usually known

before the operation. Some operators inject 2 per cent formalin into the cyst after the fluid has been aspirated. In any event, the cyst wall is incised and the membranes are extracted from within the adventitia. In a simple aseptic cyst the adventitia may be completely sutured. In most cysts, the pouch (adventitial membrane) should be marsupialized and drainage established. Septic complicated cysts may be repeatedly packed with gauze, until healing has been established.

Fig. 9.—Hooklets from scolices (Case 2).

CASE REPORTS

CASE 1.—L. C., a male student, aged 13 years, was born in the United States. At the age of 4 he visited Italy where he remained for a period of eight years, returning to this country eight months before his admission to Post-Graduate Hospital, which was Jan. 20, 1941.

For the year previous the patient had experienced pain in the left side of the chest, particularly on inspiration; in addition, he had some dyspnea on exertion. There was no cough and no expectoration; neither was there any night sweats or weight loss. There was a questionable history of hemoptysis.

The previous history was essentially negative except that one month before his admission to Post-Graduate Hospital he was a patient at a city hospital where, among other findings, the intradermal (Casoni) test

and the complement fixation (Weinberg) test were positive. The x-ray of the lungs disclosed the presence of a cyst in the left lower lobe.

The family history was entirely negative.

The physical examination was essentially negative except for dullness over the left chest, posteriorly, at about the middle two quarters. Inspiratory and expiratory râles were heard in this area. Diminished breath and voice sounds were noted.

The urine was negative as was the blood Wassermann and Kahn. There was no eosinophilia. The roentgenogram taken upon admission, Jan. 21, 1941, revealed the presence of a cystic tumor of the left posterior thorax, surmounted by a pneumothorax pocket and pneumonic reaction in the left lower lobe.

Shortly after the patient's admission to Post-Graduate Hospital he began to cough and expectorate bloody yellow mucus. At that time his temperature climbed to 104.2° F. In this connection, it must be related that shortly before the patient left the first hospital his chest had been aspirated. Feb. 17, 1941, after a few weeks of supportive treatment saddled with a little watchful waiting, the patient was operated upon. The lung was not adherent to the parietal pleura, and after segments of the left eighth and ninth ribs and the intercostal bundle were removed the wound was packed with iodoform gauze. Over this the muscles and skin were sutured. Feb. 27, 1941, the sutured incisional wound was opened and the gauze packing was removed. The lung was found to be adherent to the parietal pleura. A needle was inserted into the cyst whose fluid contents were aspirated. The overlying shell of atelectatic lung was incised, as was the adventitia. The cyst was removed. Sulfanilamide was dusted into the cavity, a Penrose drain inserted, and about it the chest wall was sutured.

The day after the first operation his temperature rose to 104.4° F. After the second stage, except for a slight chill and a low-grade fever, the patient's general condition was found to be normal (sixth day, postoperative). He did expectorate a small amount of blood-streaked mucus. March 6, 1941, one week after the operation, the sutures were removed. Occasional complaints were made of pain in the left shoulder and arm. Four days after the operation the nurse's notes read, "patient comfortable." April 1, 1941, the patient was discharged. At that time the wound was all but completely healed. For some time he had been up and walking about the ward. May 5, 1941, the records disclose the wound was completely healed. Since then he has had no complaints. At the time of this communication he is working in a machine shop.

CASE 2.—G. T., single, laborer, a 38-year-old man, was born in Syria and came to this country at the age of 10.

He was admitted to Post-Graduate Hospital Sept. 25, 1940, with complaints of inability to use his lower extremities, decrease in sensation of these extremities, and tumor in the left chest wall.

Twenty-three years before he had suffered from recurrent abscesses over the sacral and lumbar regions. They were allegedly tuberculous in nature. They did not heal for some time and then only when he exposed the parts to direct sunshine. Six years before admission a painless, barely discernible mass was noted in the chest wall on the left side, posteriorly. Three years before admission he complained of pain in this

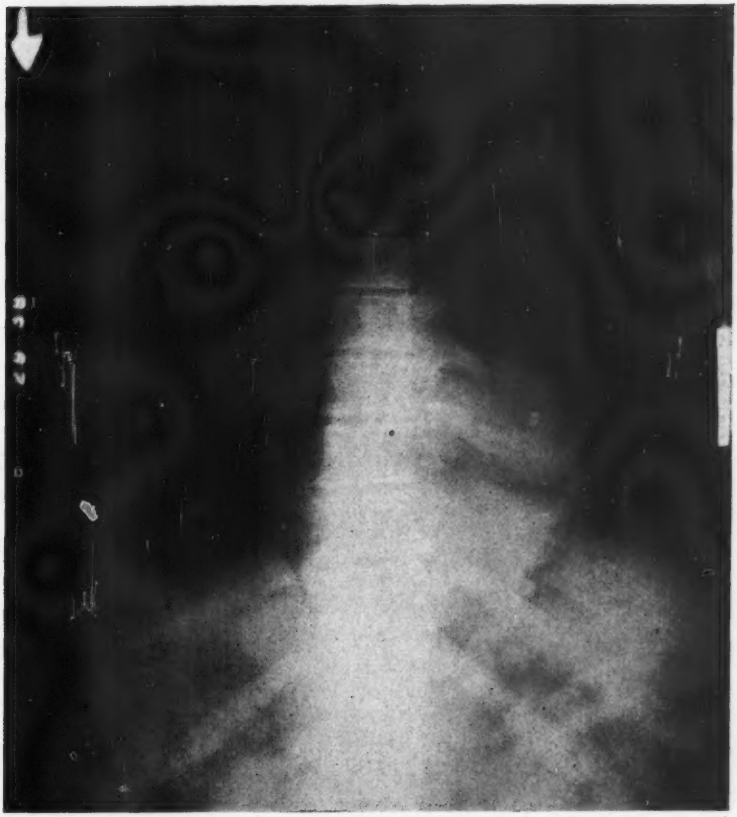

Fig. 10.—G. T. (Case 2), taken Aug. 29, 1935. Left paramediastinal mass; erosion of left ninth rib; involvement of eighth rib. (Courtesy of Dr. E. P. Pendergrass.)

area and was x-rayed by eminent Philadelphia radiographers who diagnosed his condition as a tuberculous process. Almost two months before admission he first complained of loss of sensation in the lower extremities. One morning shortly thereafter he could barely walk. The patient could not flex his legs. He said that "they were dead." There were no urinary or gastrointestinal disturbances.

The patient was a well-nourished, robust, young adult man whose gait was severely impaired. A nontender mass was noted on the left side of

the chest, mainly in the posterior axillary region extending from the sixth to the tenth ribs. Over the sacrum there are depressed pale scars. There was bilateral weakness of the legs, the left more marked than the right; the knee jerks and ankle jerks were absent. There was a bilateral Babinski sign, but no bladder or bowel disturbance. Below the fourth lumbar vertebra there was some patchy hyperesthesia. A spinal tap was

Fig. 11.—G. T. (Case 2), taken April 25, 1939, lateral view. Paramediastinal mass with vertebral body involvement. (Courtesy of Dr. E. P. Pendergrass.)

negative. The spinal Wassermann and the blood Wassermann and Kahn were negative.

X-rays revealed cystic-like degenerative changes with marked expansion involving the ninth rib on the left side and invasion of the neighboring soft tissues with encroachment on the pulmonic field with pleural retraction. There was a left paramediastinal mass. There was erosion of the seventh rib and invasion of the posterior 2 inches of the eighth rib. The lesion also invades the left transverse processes of the arches

of the seventh, eighth, and ninth dorsal vertebrae. There was no eosinophilia. A working diagnosis was made of either a benign tumor undergoing malignant changes or an extensive tuberculous process. It was decided to perform a biopsy. On September 28 the first operation was performed, consent having been obtained for a biopsy only. A tumefaction was found particularly in the area of the ninth rib over which

Fig. 12.—G. T. (Case 2), taken April 20, 1944. Same as Fig. 10, with increased costal and vertebral involvement. (Courtesy of Dr. E. P. Pendergrass.)

an incision was made. The chest wall muscles were barely opened when some fluid appeared in the wound. Upon enlarging the wound an amazing number of daughter cysts accompanied by fluid and hydatid sand were disgorged to the surface. The ninth rib was found to be destroyed, and the seventh and eighth ribs eroded. But more surprisingly, a multiloculated supra- and infracostal cyst of definite proportions was found. Aspiration of the contents was performed and the wound was packed.

October 19, the previous incision was enlarged and extended, passing through the back muscles. The cavity was ruthlessly and completely unroofed using, in the main, large biting rongeurs. After the wound was saucerized, cleaned, and painted with formaldehyde, a large leg roll was packed into it.

Fig. 13.—G. T. (Case 2), taken Mar. 11, 1944. Note disappearance of paramediastinal mass and fusion of dorsal vertebra.

One week after the biopsy the patient's temperature was 106.2° F. and three days after the operation proper, the thermometer registered 106.6° F. The pathologist reported daughter cysts, laminated connective tissue, and scolices. The specimen consisted in the main of dense fibrous scar tissue with lymphoid and plasma-cell infiltration. The hydatid fluid was bacteriologically sterile.

Fracture boards were placed beneath the patient's mattress; a Knight brace with a chin and occiput extension was worn. He did receive a short period of galvanic stimulation to the lower extremities. Postopera-

tive intradermal and complement fixation tests were negative. For some time before his discharge (Feb. 6, 1941) he had been up and walking about the ward, although he had been admitted to the hospital in a wheel chair.

Jan. 6, 1943 he was readmitted to Post-Graduate Hospital, still wearing the spinal brace. At this time he had no ankle edema although at the time of discharge this condition was present. Upon examination, one noted over the left erector spinae muscle at about the level of the

Fig. 14.—G. T. (Case 2), taken March 11, 1944, right view. Note disappearance of paramediastinal mass and fusion of dorsal vertebra.

ninth dorsal vertebra two discrete, though not too well-defined, palpable fluctuant masses. Jan. 9, 1943, these cysts were operated upon, being isolated and aspirated. The walls were removed intact. The area was packed with 10 per cent formalin gauze. About three days postoperatively this patient, who had gained considerable weight and was now definitely obese, developed a rash which was thought to have resulted from the formalin packings. These were discontinued. The x-rays disclosed much improvement, particularly good fusion of the dorsal vertebra. At the time of this communication, he was walking well, eating well, and feeling better than he had in many years. He was working as a laborer, and was not wearing the brace.

Case 3.—M. N., a white adult man, working as a ship's steward, was born in Portugal, had lived in Argentina a part of his early life. May 28, 1937, at the time of his first admission to the United States

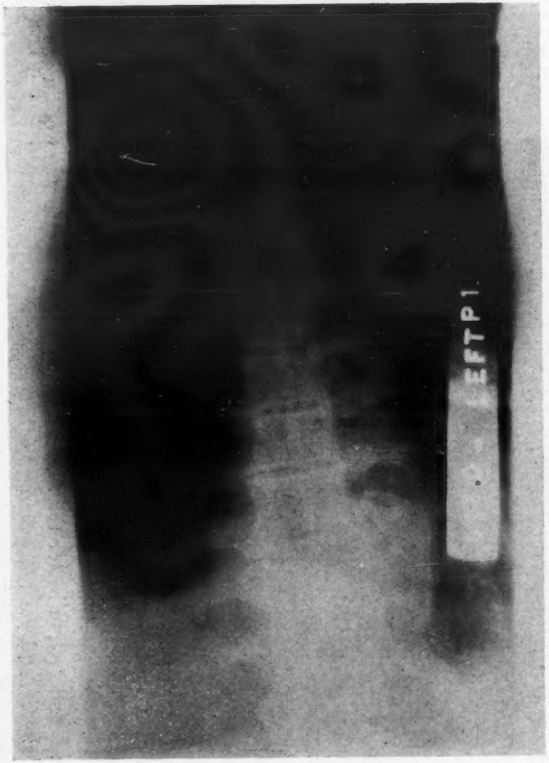

Fig. 15.

Fig. 16.

Fig. 15.—G. T. (Case 2), taken March 11, 1944, left view. Note disappearance of paramediastinal mass and fusion of dorsal vertebra.

Fig. 16.—G. T. (Case 2), taken March 11, 1944. Note disappearance of paramediastinal mass and fusion of dorsal vertebra.

Marine Hospital, Stapleton, Staten Island, N. Y., he was 35 years of age. One month before, while on board ship, he was struck by a swinging door, suffering a lacerated wound of the back. Two days later he developed pain in his left chest. On June 3, 1937, he spat blood. An x-ray picture of the left upper lobe revealed a dense circular shadow the size of a hen's egg. A diagnosis of a superior sulcus tumor was made.

Fig. 17.—M. N. (Case 3), lateral view; note perivesicular pneuma.

His second admission was March 10, 1939, when he entered the Marine Hospital for an infected right leg. Again a chest x-ray disclosed the same shadow as that noted earlier. There had been no weight loss, night sweats, fever, cough, or hemoptysis. On this occasion an additional diagnosis was made by the x-ray department; that is, a fibroma of the lung or a benign mass was injected into the picture.

A third admission was noted March 14, 1941. The patient was seen because of a blow suffered in the left temporal region. Of course, the chest mass was noted again; it was stationary and "not typical of tuberculosis." April 2, 1941, bronchoscopy, was done again and bronchograms made. All these examinations were negative.

It was learned that this patient had been admitted to Ellis Island Hospital as far back as September, 1934, at which time he had had cough, fever, and blood-tinged expectoration. At no time had the tubercle bacillus been found. The x-rays always showed the circular shadow in the apex of the left upper lobe.

His last admission into the United States Marine Hospital, Stapleton, Staten Island, was Nov. 12, 1941, at which time he was suffering from pain in the left side of the chest, hemoptysis, and cough. The

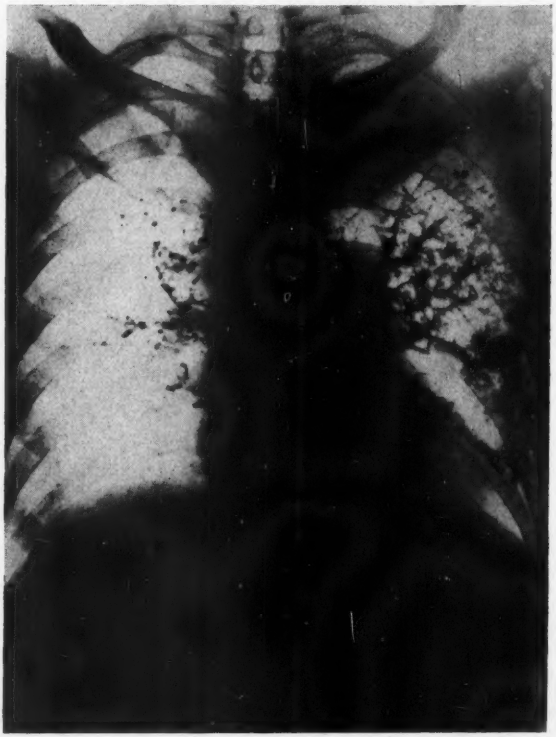

Fig. 18.—M. N. (Case 3), frontal view; again note perivesicular pneuma.

sputum was negative for tumor cells. On fourteen different occasions his sputum was negative for the tubercle bacillus. At this point I saw the patient for the first time. X-rays were presented which permitted an immediate and unequivocal diagnosis of a complicated hydatid cyst. Jan. 30, 1942, he was operated upon. A section of the left fourth rib was removed and the soft tissues were draped. The lung was adherent to the parietal pleura. The cyst was opened and suction was used. Much degenerated tissue was obtained and remnants of structures which looked like "empty grape skins" were extracted. The defect

was packed with gauze, soaked with 5 per cent formalin. Later the gauze was removed and dry gauze packing was introduced. The wound was dressed every other day. He was discharged April 16, 1942, with no cough or febrile reaction. The longitudinal incision was healed. X-rays showed complete healing of the lung. The patient gained twenty pounds in weight. The pathologic report indicated the presence of mucopurulent puttylike material. Here and there, there were cheeselike masses. There were no hooklets, laminated membrane, or scolices. The patient is at this time working as a seaman. He has not been seen in about one and one-half years.

CASE 4.*—C. L., a male child, aged 7 years, was born in the United States, although two years previous to his admission to Post-Graduate Hospital, July 31, 1935, he had visited Italy, where he lived on a farm and where he had a pet dog. His complaints were that of cough, night sweats, loss of weight, and repeated attacks of fever. There was no history of hemoptysis.

His previous illness was not significant. Physical examination revealed dullness over the left chest, posteriorly. Fremitus and voice sounds were diminished.

The laboratory tests disclosed that the urine was normal. The Wassermann test was negative, as was the Mantoux. A complete blood count was without significance, except that there was a slight anemia and there was no eosinophilia. An x-ray showed a large circumscribed area of density apparently due to encapsulated fluid in the lower lobe of the left lung. In the lower lobe of the right lung there was an area of density about once inch in diameter. The original diagnosis by the pediatric department was that of encapsulated empyema. A thoracentesis was performed and clear fluid was obtained. Because of the unusual character of the fluid, a hydatid cyst was suspected. No hooklets were found but intradermal injection of 0.2 c.c. of fluid produced a slight though definite skin reaction. In the laboratory of the Post-Graduate Hospital, the New York Presbyterian Hospital, and the United States Bureau of Public Health the complement fixation test for a hydatid cyst was performed. The results were all definitely positive. At one time the eosinophile count was 21 per cent. A diagnosis of hydatid disease was made. After the thoracic paracentesis was performed a marked pleural reaction occurred as manifested by elevated temperature and pain. A pneumothorax accompanied this and x-rays showed a thick-walled cyst in the left lung with a hydropneumothorax. There was a fluid level present in the cyst. Two weeks later the left eighth rib was removed. The cyst was thought to be central in nature and it was found inadvisable to proceed with the operation. A drainage tube was inserted into the ninth intercostal space and the chest wall was closed. The patient was discharged from

*This case was published by C. J. Leslie, Am. J. Dis. Child. 55: 1271, 1938.

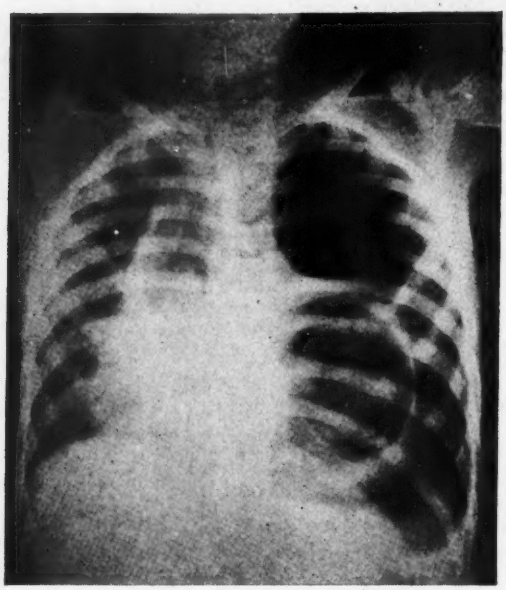

Fig. 19.—C. L. (Courtesy of C. J. Leslie. Echinococcus, Am. J. Dis. Child. 55: 1271, 1938.)

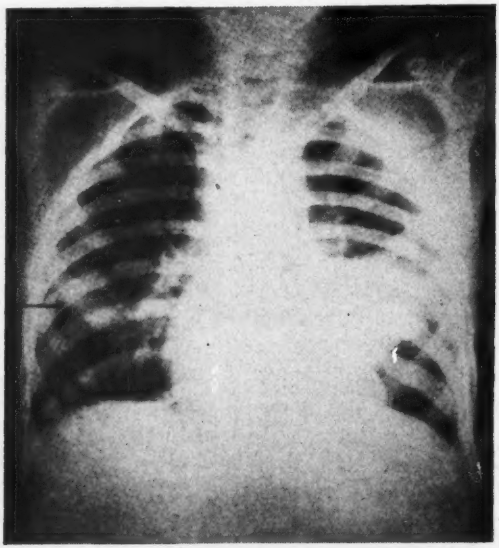

Fig. 20.—C. L. (Courtesy of C. J. Leslie. Echinococcus, Am. J. Dis. Child. 55: 1271, 1938.)

the hospital sixteen days after the operation. Jan. 2, 1936, the patient was readmitted to the hospital with an acute abdominal condition. He was operated upon and found to have a gangrenous appendix and generalized peritonitis. There was free purulent material in the peritoneal cavity and a perforation of the appendix. Intense postoperative care failed to stem the progress of the process and two weeks after the operation the patient died. Unfortunately, notwithstanding determined attempts, no autopsy was permitted. Obviously, attention would have been paid particularly to the lung.

DISCUSSION

Hydatid cysts have been found in practically every organ of the human body. Fortunately, the cyst is usually single, although on rare occasions two or more cysts are found and sometimes in one organ or part. Multiple organ infection occurs in less than 2 per cent of the cases.[4,7,8] As in bronchiectasis, so in hydatid disease, the infestation usually first manifests itself in children. In man the liver is involved in about 75 per cent of the cases; the lung is infested in about 10 per cent of the patients studied. Peculiarly, and in contradistinction to this, in the squirrel the lung is involved in 98 per cent of the cases and the liver in 0.2 per cent (Dew). This is said to be in consequence of the fact that the hepatic capillaries of this animal are readily enlarged, thus permitting the parasite to filter through. In addition, some biologic peculiarity rather than circulatory effect may influence the site of the lodgment of the embryo.

All hydatids begin as simple cysts, but in the lung when the cyst enlarges it involves a bronchus or bronchiole into which it leaks and from which the dissecting air is obtained. The contents may be evacuated through the bronchus, permitting a spontaneous cure. On the other hand, the cyst may be contaminated and all fertile elements are destroyed (Case 3). The scolices die and the membranes are destroyed.

Daughter cyst formation is rare in the lung and as already noted it is the result of some complication. In this connection, it is proper to relate that the acceptance of Case 2, where daughter cysts were found, as one originating in the lung must be questioned. Of course, superficially at least its picture is that of an intrathoracic hydatid cyst. Dew has stated that "whenever an intrathoracic cyst is found to contain daughter cysts, an hepatic origin should be suspected and this should lead to an investigation of the diaphragmatic region." We have not been able to demonstrate the presence of a cyst in the liver. However, we are not sure but that this cyst originated in the vertebral column. Possibly an implantation first appeared in the pedicle, body, or lamina of the vertebra. This presumably began when the patient was a boy in Syria. Many years later the soft tissues were invaded by extension. Just before he was seen by me, cyst formation within the spinal canal reached such a stage that pressure was exerted upon the

cord and concordant symptoms appeared. This, of necessity, is a very slow process because decompression probably had occurred time and again by extraspinal extension. Of course, the accuracy of the observations made at the time of the operation may be questioned. The history, the findings, and the postoperative neurologic improvement must permit of the biting inference that this was not a pulmonary hydatid but rather it was a hydatid cyst originating in the spinal column.

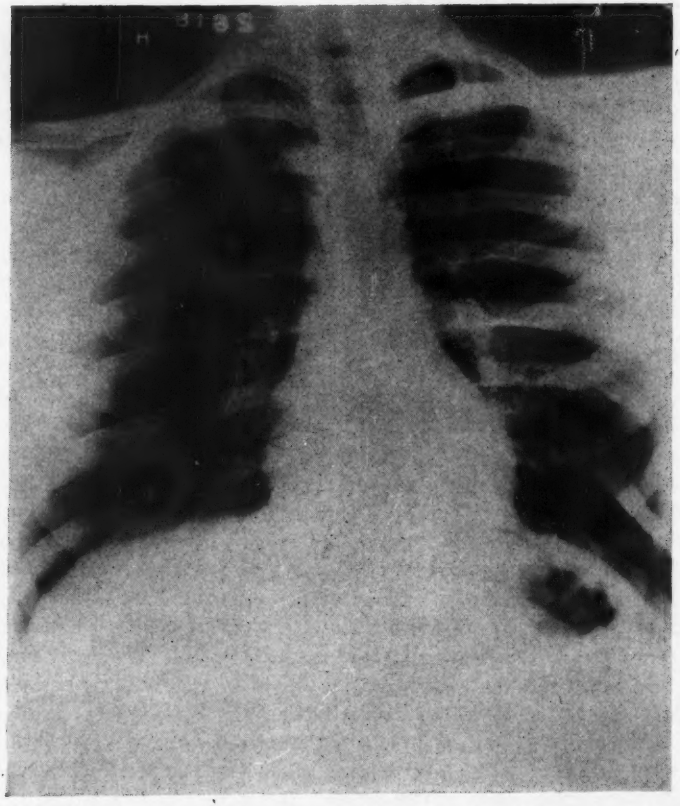

Fig. 21.—Roentgenogram taken April 8, 1944 (L. C.) cyst removed (note Fig. 6) from left lower lobe.

Again, according to Dew, "the prognosis in vertebral hydatid disease is almost hopeless as regards complete cure. This is due to the impossibility of removing by surgical means all diseased bone . . . to the multiplicity of extraosseous cysts, to the certainty of recurrent pressure on the cord and to the risks of infection." It is also noted in this case that at the time of the operation when unsuspected scolex containing fluid was evacuated, these premature heads were undoubt-

edly implanted in the operative wound thereby permitting of the growth of a recurrent or a secondary hydatid cyst.

Almost all authorities deprecate the aspiration of the hydatid cyst. In many instances this procedure has been followed by death. The patients in Cases 1 and 4 were obviously quite fortunate, since they were aspirated prior to the time of the operation. Even if death does not result, infection of the cyst and/or contamination of the pleural cavity are probabilities. Even if the foregoing do not occur there is

Fig. 22.—Lateral view taken April 8, 1944 (L. C.).

always the possibility of creating the presence of a lifeless foreign body in the lung. In fact, the death of the parasite does not remove the patient from the possibilities of danger. Finally, let it be said, it takes many years to learn whether leakage around a puncture needle occurred with subsequent implantation of scolices in the chest wall.

It is not unusual to have a simple cyst leak and rupture into a bronchus or bronchiole. If this progresses the fluid and the membranes

may be extruded, permitting a spontaneous cure. If only the fluid is expelled, the membranes remain and one may expect an infection or abscess formation. Paradoxically, these complicated cysts may possess one virtue, that is, a pathognomonic radiograph may be obtained. In Case 3 a preoperative diagnosis was made in view of the presence of a perivesicular pneuma. This feature has been most adequately described by Arce and Zehbe.[492]

There is but one treatment of hydatid cysts and that is surgical. The absence or presence of adhesions modifies the plan of attack. If there are definite adhesions the rib overlying the cavity is removed and the contents are in turn extracted (Case 3).

Most authorities insist, particularly with a simple cyst, that one should never feel that the adhesions are either quantitatively or qualitatively what one desires. Therefore the operation should be performed in either two stages or, if in one stage, the lung should not be permitted to retract into the thoracic cage. This is prevented by using increased anesthetic gas or oxygen pressure; or the lung outside of the cystic area should, as a preliminary, be sutured to the chest wall. It may be indicated that Arce suggests preoperative pneumothorax for the determination of the presence of and the location of adhesions. He also uses it for operative convenience. I may possess a clouded conception of this disease, yet, being what it is, I must look upon the utilization of pneumothorax either diagnostically or operatively as a dangerous procedure. In making this statement, I am fully aware of the fact that no surgeon in the United States should border on such tactlessness by virtue of his limited experience. However, although my approach may appear presumptuous, I desire no classification in such a category. My conclusions are based upon the fact that simple peripheral hydatid cysts may be ruptured by a pneumothorax refill, just as a putrid lung abscess which also is a peripheral process may rupture into a pleural space with the use of pneumothorax therapy. Further, if the lung is not adherent to the chest wall, an incision when made through the thoracic cage permits of the collapse of the lung. Finally, the localization is very well noted by a spot-shot. It follows then that my impression is that a pulmonary hydatid cyst should be operated upon in two stages or in one stage using increased anesthetic pressure of 10 cm. of water when the cyst is approached.

Roberto Estevez[181] discusses the injection of 2 per cent formalin into the cavity after it has been punctured and aspirated. Dévé most emphatically insists that this medicament should never be used in the lung or pleura. Of course, under all circumstances both the incisional wound and the pleural cavity should be well protected during the operation. The aspirating unit must be functioning and immediately at hand. Fluid is not always obtained from a complicated cyst (Case 3). After the cyst has been removed the adventitia may be closed

tightly or it may be sutured to the chest wall bringing a drain from the pulmonary defect (marsupialization).

Biologic reactions are encountered in many ways. As in all helminthic diseases an eosinophilia is noted (Case 4). This is not a constant finding. The Weinberg test, which has been confirmed, is a complement fixation test. One must possess a fresh antigen properly tested. Although more reliable than the leucocyte count, it too is not always reliable. The best type of test is that which involves an intradermal reaction and is known as the Casoni test. A positive reaction (like a Mantoux test) presents a wheal and a surrounding area of erythema.

The diagnosis of pulmonary hydatid cyst is difficult, particularly in the United States where it is a rare condition. Since this cyst becomes complicated quite often certain differences arise which add to the problem. In brief, one may be suspicious of a case if there is a history of cough, pleural pain, hemoptysis, and/or a febrile reaction, particularly if the patient has been an inhabitant of some infested area. X-rays may be of great help and other laboratory findings as indicated may be helpful. Cysts usually are in the lower lobe and occur more frequently in males.

SUMMARY

Up until 1930, only thirty-six cases of pulmonary hydatid cyst had been reported in the United States. Only two of these patients were born in the United States.[493] Four additional patients with pulmonary hydatid cyst are reported, all of whom came to surgery. The sundry manifestations including the pathognomonic radiographic findings have been discussed. The sign permitted a preoperative diagnosis. The rationale of treatment has been touched upon. This is dependent upon the type of cyst: central or peripheral, simple or complicated. The disease will probably increase with the returned soldier.

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DISCUSSION

DR. LEO ELOESSER, San Francisco, Calif.—In Argentina and Uruguay these hydatid diseases are daily bread. The South American literature, as Dr. Davidson has intimated, is full of discussions as to the methods of treatment, whether to marsupialize or not, and how to treat the primary cyst. In the best of events, whether marsupialized or not, the treatment is full of complications and is long-lasting and difficult. I think very likely that as the South American clinics are provided with good anesthetists all of these methods will be supplanted by lobectomy of the lobe containing the cyst which should vastly shorten the time of convalescence and vastly diminish complications.

DR. CAMERON HAIGHT, Ann Arbor, Mich.—I wish to elaborate on only one point, namely, the incidence of hydatid disease of the lung on this continent. About four years ago Dr. Alexander and I reviewed the cases that had been reported in Canada and the United States. At that time we were able to find in the literature only forty-four instances of hydatids of the lung or pleura in North America. We reported two cases, both patients being immigrants. Of the total of forty-six cases, only five of the patients were born in the United States and of these, only two had never been out of this country. One patient was born in Virginia, but had been in Europe for a period. In this case it is believed that the disease was acquired in this country, whereas the source of infection in the remaining two of the five patients is not known. The point to be stressed is that almost all of the patients with hydatid disease who have been seen, to date, in the United States or Canada have been immigrants.

We also reviewed the patients with hydatid disease of other organs who were seen at the University Hospital during the period covered by our report. There were six instances of hydatids of the abdominal cavity, and in all of these cases the disease had occurred in patients who were not American-born.

DR. A. R. VALLE, Buenos Aires, Argentina.—I would like to say a few words because I am from Buenos Aires, Argentina, where we see many cases of hydatid cyst of the lung. First, I would like to congratulate Dr. Davidson for his excellent paper as I am surprised to find that some one here is so familiar with a disease that does not frequently occur in this country.

We have found that the Casoni test is very reliable in about 70 per cent of the cases. We almost never use the complement fixation test or the Ghedini test.

In regard to the x-ray studies, we found that the most conspicuous feature was the rounded, homogeneous, and well-defined shadow. We do not consider of great importance, the semicircular shadow, so-called pathognomonic by some South American writers, because we did not find it present as often as they claim.

Incidentally, in Dr. Graham's service a few months ago I saw that so-called semicircular shadow in a proved case of bronchiogenic carcinoma.

In regard to treatment of the disease, I am sorry that Dr. Alejandro Ceballos' work was not mentioned. I believe that in Argentina he has had the most experience with hydatid cysts of the lung. He has published an article in the JOURNAL on the technique of operation for hydatid cyst of the lung. He advises the removal of the uncomplicated cyst of the lung in one stage, with which procedure I agree. I see no reason to perform the operation in two stages if the parasite is alive and not infected.

In this condition, we do not use preoperative pneumothorax, which, unfortunately, is very popular in this country. We believe it complicates the condition and also is dangerous, as I have seen several patients in which pneumothorax, as a diagnostic procedure, broke open the cyst, thus causing an allergic reaction and respiratory distress which in some cases were fatal.

DR. WILLARD VAN HAZEL, Chicago, Ill.—I would like to add one case which was very interesting, being complicated by a communication with the liver. A Grecian woman was first operated upon because of costal margin pain with a mass which was thought to be gall bladder, but the gall bladder was normal though the liver was enlarged. Because of productive sputum and cough and a small amount of hemoptysis, we were asked to see her. There was a shadow above the diaphragm on the right side. We felt that we were dealing with an abscess without a fluid level as is often true, and we explored this region. Adhesions were present at operation and the abscess was entered after aspiration, many small cysts being removed. We could then put our finger—in fact, we could put two fingers—down through the diaphragm into the liver, and we brought out innumerable cysts of all sizes which proved to be echinoccocus. I would estimate that this cavity in the liver was at least the size of a good-sized orange. Both the liver abscess and pulmonary abscess drained through this opening and obliterated themselves.

LIEUTENANT COLONEL RICHARD H. MEADE, JR., Memphis, Tenn.—About ten years ago, Dr. Ravdin, at the University of Pennsylvania Hospital, saw a very interesting echinococcus cyst of the mediastinum which was operated on with the erroneous diagnosis of pulmonary cyst, or pleural effusion. I report this mainly to ask the question about the frequency of allergic reactions. The cyst was opened inadvertently, and its contents spilled into the pleural cavity. It was only when the daughter cysts were found, that the diagnosis was made. The cyst was enucleated, as I remember it.

The patient made an uneventful recovery and the last I heard, which was only eight months afterward, he had returned to the Pennsylvania coal fields. I don't know anything about the subsequent history. I have been impressed from what I have read about the dangers of spilling the fluid into the uninvolved pleural cavity.

DR. LOUIS R. DAVIDSON, New York, N. Y.—If I have time, I shall show the lantern slides. Primarily, I should like to answer the questions which have been propounded. In my paper, I could not touch upon, completely, the question of surgical treatment. There are four distinct methods of handling hydatid cysts of the lung which are totally dependent upon the disposal of the cyst and the treatment of the adventitia.

First, the so-called Posodas method: In simple cases, one may suture the edges of the adventitia. This method has already been touched upon.

The next procedure which, I believe, has been abandoned even by South American surgeons is that of harpooning. When the cyst is delivered through the chest wall wound, sutures are passed through the chest wall and the lung outside

of the area of the cyst; thus, the cyst is held to the chest wall. Subsequently, the cyst and the membranes are suitably and adequately taken care of.

Third, as to pneumothorax therapy in the treatment of the cyst, I should like to read a sentence which appears in my paper, "I must look upon utilization of pneumothorax either diagnostically or operatively as a dangerous procedure." In making this statement, I am fully aware of the fact that no surgeon in the United States should border on such a premise, since none of us has had a tremendous experience with this particular lesion. The method is dangerous and Dr. Valle has already told you that spillage may occur after performance of a pneumothorax.

This leads to the question which has been propounded by Dr. Meade. However, before I answer that, I should like to say the fourth and final method is that of complete excision, which in the case of hydatids of the lung would mean either lobectomy or pneumonectomy.

In one of the cases which I have reported, and in which, I believe, a cyst arose in the vertebral column, I did not anticipate at the time of operation finding a hydatid cyst. At the time of operation, when a chest wall incision was made, some fluid appeared. Students happened to be in the operating amphitheatre. I turned to them and said, "We have a cyst here." I meant every cyst in the world but a hydatid cyst. Thereopen, the incision was enlarged and much to my consternation, hundreds and hundreds of small grape-sized daughter cysts poured out, obviously, accompanied by innumerable, all but microscopic daughter cysts. Daughter cysts, only very infrequently, appear in the lung. When they appear elsewhere in the body they are the result of mechanical or toxic injury to the germinal layer. That is how they develop. One year later, I again operated upon this man. He was re-operated upon because originally I did not protect the operative field.

This condition is known as secondary hydatidosis. Therefore, when one operates for a hydatid cyst, he should drape the field very carefully and he should have an aspirating syringe in the incisional wound.

Regarding the question raised by Dr. Eloesser, I can say, from a very limited experience, that in the very great majority of cases, a lobectomy is unnecessary, since we would be divorcing the patient of a certain amount of his vital capacity. The youngster, L. C., at the age of 8 years, returned to Italy and lived there for eight years. He was operated upon in two stages. If I were to take over a similar case at this time, I should perhaps perform the operation in one stage. This patient is now 16 years of age and is very powerfully built. He is working at a very strenuous job as a mill hand.

The patient who had daughter cysts is now working as a laborer.

The patient whose x-rays revealed a pathognomonic, perivascular pneuma is again working as a seaman.

I feel perhaps that if we can remove the hydatid cyst, it is better than removing the lobe in which it is found.

Emile Holman recently touched on the question of cysts found in individuals born in this country and who never left the United States.

I am conversant with the work of Ceballos. His is a very interesting paper.

To touch upon the matter propounded by Dr. Valle: Every case does not show a characteristic x-ray sign. It is noted only when the cyst becomes complicated, and it is complicated only when the adventitia is involved. If one is fortunate enough to see the patient directly after air has seeped between the laminated layer and the adventitia, then the diagnosis is made and is absolute.

SURGICAL LIGATION OF A PATENT DUCTUS ARTERIOSUS ASSOCIATED WITH AN ANEURYSM OF THE PULMONARY ARTERY

Report of a Successful Case

James W. Nixon, M.D.
San Antonio, Texas

A STUDY of the literature fails to reveal a case of aneurysm of the pulmonary artery associated with a patent ductus arteriosus which has been successfully treated surgically. Thus it is felt that such a case, recently encountered, is of sufficient interest to warrant reporting.

Although patent ductus arteriosus, next to patent foramen ovale, is considered the most common congenital cardiac defect, aneurysm of the pulmonary artery associated with a patent ductus is rare. In 1924, Moench[3] reported a case of aneurysmal dilatation of the pulmonary artery with patent ductus arteriosus in which death resulted from rupture of the aneurysm into the pericardial sac. He states that this case was the first encountered on the record of the Office of the Chief Medical Examiner of the City of New York since January 1, 1918, during which time 65,000 cases with almost 10,000 necropsies had been handled. Boyd and McGavack,[7] in a review of the literature of all cases of aneurysm of the pulmonary artery since 1833 up to 1939, present only 111 examples of such aneurysms in which the diagnosis was confirmed in each case by necropsy. Of this group, congenital anomalies were present in 66 per cent of the cases, and were considered etiologic factors in 43.2 per cent. Open ductus arteriosus occurred in 23 per cent. Boyd and McGavack observed that although pulmonary hypertension occurs in cases of patent ductus arteriosus and has been regarded as causative of aneurysm, patency of this passage is not uncommon without aneurysm, and accordingly it seems reasonable to assume that some additional lesion may contribute to the production of aneurysm in this group. Moench[3] stated that the most important factor as far as etiology of pulmonary aneurysm is concerned seems to be a patent arterial duct and that a number of cases reported by Henschen[9] and Posselt[10] showed such a condition. Syphilis also has been considered an important causative agent;[7, 9, 11, 12] however, in our case syphilis was not contributory.

Although a true aneurysm of the pulmonary artery is rare, dilatation and thinning of the pulmonary artery may be present in cases of patent ductus arteriosus. Gebauer and Nichol,[14] and Touroff[15] have observed that this feature was most marked in the oldest patients. This is one reason it is felt that surgical closure should be performed as soon as

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the presence of a patent ductus is definitely determined, providing of course, that there is no contraindication to operation. Touroff[15] states that excluding the factor of the patient's age, the degree of dilatation and thinning of the pulmonary artery appear to be proportional to the diameter of the ductus and the completeness of ductal patency.

As is generally known, in fetal life the ductus arteriosus serves to divert the blood from the pulmonic artery to the aorta, thus by-passing the lungs. When the child is born and the lungs expand, the ductus normally closes and the blood in the pulmonary artery passes through the lungs to be aerated. The failure of the ductus to close results in a reversal of flow, and blood passes to the pulmonary artery from the aorta through the ductus. According to Gross[2] probably the most important factor in the etiology of a patent ductus is the position and direction of the vessel. The duct usually enters the aorta at an angle, and as the acuteness of the angle decreases, the ductus becomes more and more exposed to intra-aortic pressure. If the angle becomes a right or an obtuse angle, the force of intra-aortic pressure can readily be exerted to maintain patency of the lumen of the ductus. It is logical to assume that the greater the degree of patency of the ductus, the greater would be the pressure and volume of aortic blood passing into the pulmonary artery through the duct. The much thinner wall of the pulmonary artery, which is not intended for such pressure, then would tend to yield to form an aneurysm. As observed by Mackler and Graham[16] the basis for aneurysmal dilatation is established with the persistence of a patent ductus. In cases of aneurysm of the pulmonary artery associated with a patent ductus, the most commonly proposed explanation in addition to that of increased intrapulmonary arterial pressure, is the weakening of the wall of the artery at the site where the aortic blood strikes it as it flows through the patent ductus.
Gross[5] has stated that recognition of the possibility of an aneurysm either of the ductus itself or of the pulmonary artery as a complication of a patent ductus not only serves to emphasize the diagnostic confusion which may arise, but adds another seldom mentioned argument in favor of surgical closure of recognized patent ductus.

REPORT OF CASE

The patient, N. G., a white girl aged 19 years, was admitted to the Santa Rosa Hospital, Jan. 28, 1944. She had consulted her physician because of dyspnea on exertion, and the diagnosis was made of patent ductus arteriosus.

Past History.—The patient's previous medical history was negative except for a humming and roaring in her chest present since early childhood. This sound became more pronounced when she lay down. Her mother stated that when she slept with the patient she could hear the unusual sound of the child's heart even in infancy. The patient

had suffered no attack of acute infection or serious illness. Although during childhood she had been rather frail, in recent years she had gained considerable weight. She complained of tiring easily and of a pain over her heart when tired. She reported that after roller skating for ten to fifteen minutes she was forced to rest because of dyspnea and palpation.

Physical Examination.—The girl was apparently in good health and was exceptionally well nourished. There was nothing remarkable about her physical examination except for her heart, which had a definite machine murmur that was transmitted to the left axilla and to the back. A pronounced thrill could be felt over the pericardial region.

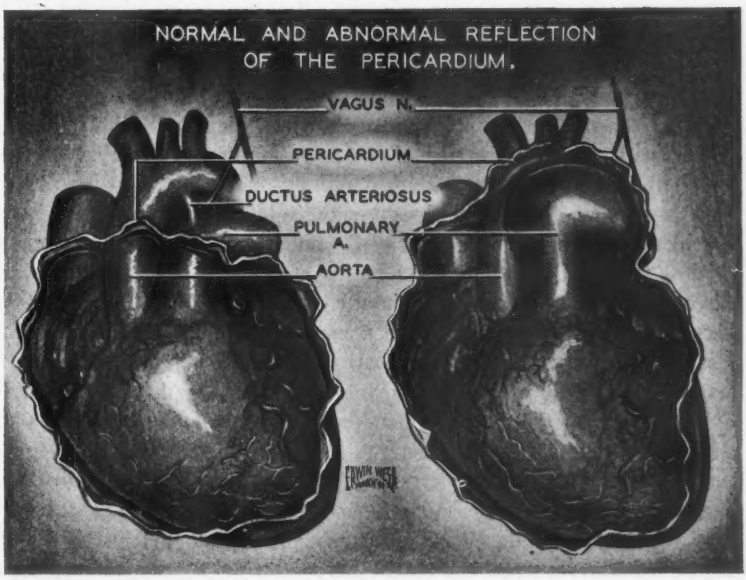

Fig. 1.—*A.* Position of pericardium as it is normally reflected from the pulmonary artery. *B.* Pericardium reflected from the aorta as found in this case.

Laboratory Findings.—The urine test was entirely negative except for a trace of albumen. The blood Wassermann test proved negative. The red blood cell count was 5,090,000, the white blood cell count, 9,950; the differential showed 71 per cent polymorphonuclears, 26 per cent lymphocytes, 1 eosinophile, 2 mononuclears, and 4 stab forms. The hemoglobin was 94 per cent, the coagulation rate, 4.5 minutes. The blood pressure was 112/60. The electrocardiogram was normal.

Operation.—The patient was operated upon Jan. 29, 1944, under intratracheal cyclopropane anesthesia. The usual anterior incision for patent ductus arteriosus was made over the left second intercostal space.

On opening the pleural cavity, we were immediately confronted with an expansile, pulsating mass which we had not encountered in any of the other similar operations for patent ductus. The mass was definitely within the pericardium and extended upward in the region of the aorta. An incision was made through the mediastinal pleura above the pulsating mass with the idea of exposing the ductus. It was found that the pericardium, instead of being attached to the pulmonary artery as is normally the case, was attached to the inferior surface of the aorta, thus overlying the ductus, and necessitating an incision through the pericardium. When the pericardium was opened, the mass was revealed to be an aneurysm of the pulmonary artery. It was readily discovered that the aneurysm lay anterior to the ductus arteriosus, completely obscuring it. By retracting the aneurysm downward, the ductus was exposed, and in the usual manner freed from its surrounding structures. The ductus entered the aorta at a marked obtuse angle, which was undoubtedly an explanatory factor in the patency of the ductus. The ductus was ligated with No. 8 braided silk, following which the thrill disappeared and the pulsation of the aneurysm diminished to a marked degree, thus definitely indicating that the blood flowing through the patent ductus with its aortic pressure was exerting increased strain on the wall of the aneurysm. No effort was made to deal with the aneurysm. By means of a sterile stethoscope placed directly over the heart, it was observed that the machine murmur had been converted to the sounds of a normal heart. The pericardium was closed with interrupted cotton sutures. The ribs were held in normal position by means of No. 30 wire sutures, and the muscles and skin were approximated with continuous locked sutures. The patient had a rapid recovery and was discharged from the hospital on the eighth postoperative day. Three weeks after the operation, dyspnea had greatly improved and she had returned to work as a clerk at an army field. The patient developed a hoarse voice postoperatively, due to injury of the recurrent laryngeal nerve during the rather extensive dissection which was carried out in an effort to locate the ductus, but within five weeks her voice had become normal.

COMMENT

This case was not one of simple uniform dilatation of the pulmonary artery, for the artery abruptly dilated and formed an enlarged pulsating mass with a very thin wall. On each side of the aneurysm the pulmonary artery retained its normal diameter. It would seem, therefore, that this case must be considered as a true aneurysm of the pulmonary artery associated with a patent ductus arteriosus. It is believed that the possible cause of the aneurysm was the abnormal position of the ductus in its relationship to the aorta.

SUMMARY

Review of the literature indicates the rarity of cases of aneurysm of pulmonary artery associated with patent ductus arteriosus.

Possible causative factors of pulmonary aneurysm are mentioned with special attention to the relationship of a patent ductus.

Report is presented of a successful surgical ligation of a patent ductus arteriosus associated with an aneurysm of the pulmonary artery.

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TRANSTHORACIC GASTRIC SURGERY WITH A REPORT OF A TRANSTHORACIC RESECTION OF A DIVERTICULUM OF THE STOMACH

MAJOR DONALD L. PAULSON, MEDICAL CORPS, U. S. ARMY

TRANSTHORACIC gastric surgery constitutes a relatively new approach to the surgical treatment of gastric lesions. It received its impetus from attempts to relieve malignant obstruction at the esophageal outlet, whether the tumor was on the gastric or esophageal side. In 1938, Adams and Phemister[1] reported the first successful one-stage transthoracic resection and anastomosis of the esophagus and stomach for a primary carcinoma of the lower end of the esophagus. In the same year, Ochsner and DeBakey[2] performed the first successful transthoracic resection and anastomosis for a carcinoma of the cardiac end of the stomach. Since that time Garlock,[3] Churchill and Sweet,[4] Clagett,[5] Holman and McSwain,[6] Stephens,[7] and Raven[8] have performed several successful transthoracic resections with intrathoracic esophagogastrostomies for carcinoma of the distal end of the esophagus or cardiac end of the stomach. While the details of the surgical technique are still in the developmental stage, it is safe to state that the transthoracic approach is the operation of choice for obstruction at the esophageal outlet whether the tumor is primarily in the stomach or in the esophagus.

Willy Meyer,[9] Churchill and Sweet,[4] Sweet,[10] Pack and McNeer,[11] and others have applied still further this newer concept of the surgical approach to the stomach and have reported total gastrectomies with the construction of an intrathoracic esophagojejunostomy accomplished through the transthoracic route. In some cases the entire stomach, the regional lymph nodes, and the spleen and tip of the pancreas were removed as one block of tissue (Sweet,[10] Churchill and Sweet[4]). These cases illustrate the technical feasibility of total gastrectomy through a transthoracic approach. In view of the favorable results that have been obtained with total gastrectomy by the abdominal approach, the employment of the transthoracic approach for cancer of the upper portion of the stomach which does not obstruct the esophagus may be open to question. As pointed out by Churchill and Sweet,[4] and by Clagett,[5] the transthoracic approach has the advantage that it permits resection of a greater portion of the lower esophagus than does the transabdominal approach. Carcinoma of the cardiac end of the stomach frequently extends microscopically through the submucosa into the esophagus for varying distances. The transthoracic approach allows freer access to and more extensive resection of the lower esophagus so that there is less possibility of leaving behind carcinoma in that organ. Total gastrectomy may be avoided in some patients in

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whom the carcinoma is high in the cardia if the transthoracic approach is utilized, whereas total gastrectomy would be mandatory if a transabdominal approach be employed.

Beecher[12] recently compared the shock-producing effects of the transabdominal approach to gastric carcinoma with the transthoracic approach. Seventeen patients who had undergone gastric resection through the thorax were compared with seventeen who had undergone gastric resection through the traditional transabdominal route. The patients in both groups were operated upon under ether anesthesia. He concluded that the transthoracic approach produced no more shock, and perhaps even less, than did the transabdominal approach to the stomach. Beecher explained this on the basis that gastric surgery through the thorax can be carried out at a lighter level of anesthesia than is possible when the transabdominal route is used. In using the transthoracic approach, relaxation of the abdominal wall is not necessary. The only relaxation needed is that of the diaphragm, and this can be obtained by blocking the phrenic nerve by either crushing it or injecting it with procaine. Second, Beecher believes that when the transabdominal route is used, the surgeon is constantly putting pressure or tension on the large blood vessels and nerve plexuses of the abdominal cavity, the effect of which is to produce a fall in blood pressure. In the transthoracic approach there is little disturbance of the intra-abdominal structures adjacent to the stomach because of adequate exposure. Although adequate data have not been accumulated, Beecher states that there is reason to believe that gastric surgery carried out through the thorax with light anesthesia and without an abdominal incision is followed by a lower incidence of postoperative pulmonary complications than is true following the transabdominal approach. Churchill and Sweet[4] concur in this opinion. There is no splinting of the abdominal muscles postoperatively when the transthoracic approach is used, and the patient is able to cough more effectively. Added to the advantages of less shock and possibly fewer postoperative pulmonary complications is the advantage of adequate anatomic exposure obtained by the transthoracic approach. The successful performance of transthoracic gastric surgery presupposes a properly qualified surgical personnel including a well-trained anesthetist experienced in the proper management of thoracic surgical cases.

The transthoracic approach has been applied mainly for malignant lesions of the stomach and esophagus. The following case report illustrates its application for a benign lesion, namely a diverticulum of the cardiac end of the stomach.

CASE REPORT

A 26-year-old officer was transferred to the hospital with a diagnosis of diverticulum of the stomach. Two months previously he had noted loss of appetite, a feeling of fullness after eating, aching epigastric and generalized abdominal pain unrelated to meals, nausea, and vom-

iting. The feeling of fullness was accentuated by lying down and relieved by walking about or by vomiting. Vomiting did not relieve the pain, however. The vomitus usually consisted of only a small amount of undigested food mixed with gastric juice. There was no hematemesis nor melena. Stomach x-ray at another hospital revealed a diverticulum of the cardiac end of the stomach. He was given a soft, bland diet following which his symptoms were but slightly improved. His appetite continued to be poor, and he had lost about twenty pounds since the onset of illness, two months previously.

Physical examination revealed a well-developed, undernourished man of an asthenic habitus. There was slight tenderness on deep palpation in the epigastrium, but no masses were palpable. In conformity with his asthenic habitus the anterior costal angle was small, and the costal margin extended well down over the abdominal region. The remainder of the physical examination was negative.

Laboratory findings including blood counts, blood serology, blood urea, serum proteins, and gastric acids were all within normal limits. Numerous stool specimens were negative for occult blood.

X-rays of the esophagus, stomach, and duodenum revealed the presence of a diverticulum 4 cm. in length located near the lesser curvature of the stomach arising from the posterior wall just below the esophagogastric junction. There was no evidence of herniation through the diaphragm, and the remainder of the stomach and duodenum appeared normal. Roentgenogram of the chest was negative, and the Graham-Cole test revealed a normally functioning gall bladder.

Gastroscopy revealed an ostium, 0.5 cm. in diameter, assumed to be the opening into the diverticulum, in the posterior wall of the stomach within 2 cm. of the esophagogastric junction, near the lesser curvature.

The diverticulum of the cardiac end of the stomach was believed to account for the patient's symptoms. Surgical removal of the diverticulum was recommended. Because of the location of the diverticulum in an individual of an asthenic habitus, it seemed likely that the transabdominal approach would be extremely difficult because of inadequate exposure. The transthoracic approach was considered preferable because of the technical ease of approaching the diverticulum. Accordingly, through a transthoracic, transdiaphragmatic approach, the diverticulum of the cardiac end of the stomach was excised. Nitrous oxide, oxygen, and ether were administered through an intratracheal tube. A posterolateral skin incision was made along the course of the left seventh rib. After dividing the latissimus dorsi, the trapezius, and a few fibers of the serratus anterior muscles, the seventh rib was resected subperiosteally from the angle of the rib to the costochondral junction. The pleura was incised and no adhesions were encountered. The pulmonary ligament was ligated and divided. The phrenic nerve was lightly crushed to paralyze the diaphragm. The diaphragm was then incised anterolaterally for a distance of 6 cm. beginning 2 cm.

anterolateral to the esophageal hiatus. The peritoneal reflection from the fundus of the stomach to the diaphragm was divided. The diverticulum was easily found lying posteriorly, originating midway between the greater and lesser curvatures of the stomach. The diverticulum and adjoining portion of the stomach were drawn up through the opening in the diaphragm. A straight forcep was used to clamp the wide base of the diverticulum which was then removed by surgical diathermy and the base coagulated. The defect in the stomach was closed by a Parker-Kerr stitch of fine chromic catgut placed over the forcep to invert the base of the diverticulum. A second row of fine chromic catgut used as a running suture, and a third row of interrupted fine silk sutures completed the closure. A very easy and satisfactory closure was obtained. There was no soiling. The stomach was replaced into the peritoneal cavity, and 3 Gm. of sulfanilamide crystals were sprinkled in the peritoneal cavity. The incision in the diaphragm was closed by interrupted mattress sutures of braided silk so placed as to overlap the margins of the incision. A running suture of fine chromic catgut was placed along the free edge of the overlapped diaphragm to approximate the pleura. The pleural cavity was irrigated with sterile water. A catheter was placed in the pleural cavity by means of a trocar in the eighth intercostal space in the posterior axillary line and connected to a water seal system. The pleura, muscles, and skin were closed in layers. At the completion of the operation bronchoscopy was done and two ounces of mucoid material were aspirated. X-ray pictures of the chest taken immediately following bronchoscopy revealed both lung fields to be clear, with complete expansion of the left lung.

The patient's condition was good throughout the operation. At no time did his blood pressure drop below 120 mm. of mercury, systolic, and 60, diastolic. His pulse rate did not exceed 100 per minute. He was given 1,000 c.c. of 5 per cent glucose in saline, 500 c.c. of plasma, and a 500 c.c. blood transfusion during the operative procedure.

The diverticulum was reported by the pathologist to consist of gastric mucosa, a thin muscular layer, and a thick outer fibrous layer.

The postoperative course was uneventful. The catheter was removed from the pleural cavity on the third postoperative day. The patient was out of bed on the seventh postoperative day. Five weeks after the operation the officer was given a thirty-day sick leave. On his return, roentgenograms of the chest revealed the lung fields to be clear with flattening of the left diaphragm and obliteration of the left costophrenic angle. Stomach x-rays revealed no abnormalities except for a moderate hypertrophy of the gastric mucosa. He had no further symptoms and was returned to duty eleven weeks after the operation.

DISCUSSION

The case presented here illustrates the application of the advantages of the transthoracic approach for the resection of a benign lesion of the

cardiac end of the stomach. The exposure of the gastric diverticulum situated high on the posterior surface of the stomach near the junction with the esophagus, in a patient of an asthenic habitus with a narrow costal angle, would have been difficult through an abdominal approach. Through a transthoracic approach the diverticulum was resected with ease and the base of the diverticulum closed satisfactorily without soiling. The advantages of the transthoracic approach to gastric surgery as compared to the transabdominal approach are: (1) adequate exposure, (2) lighter anesthesia, (3) less traction or pressure on the larger vessels and nerve plexuses near the stomach, and (4) possibly fewer postoperative pulmonary complications.

In the case of malignant lesions of the cardiac end of the stomach, the transthoracic approach affords freer access to, and more extensive resection of, the frequently involved lower esophagus than does the transabdominal approach. Total gastrectomy may be avoided in some cases because of the technical ease of performing a transthoracic esophagogastrostomy.

The advantages of the transthoracic approach to the upper abdomen may also be applied to major thoracico-abdominal war wounds. It is apparent that if the chest is opened as a result of a major chest injury, the abdomen may be explored through the diaphragm and appropriate procedures carried out through the same incision, thus obviating a second abdominal incision in some cases.

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